Muscle Forces and their Contributions to Acceleration during Stair Descent

Undergraduate Thesis

In Partial Fulfillment of the Requirements for Graduation with Research Distinction in Mechanical Engineering at The Ohio State University

By

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Abstract

Stair descent (SD) is a difficult, potentially dangerous task for populations with lower limb pathologies, such as the elderly and those with osteoarthritis (OA). Previous studies have investigated lower limb joint torques and muscle activations during SD in young, healthy populations using traditional gait analysis. These results have been used to inform rehabilitation for pathological populations, which often focuses on muscles that are active during SD. However, rehabilitation is not 100% effective. Other studies have also examined how speed affects SD since young, healthy populations often perform SD at faster speeds than those with pathologies. At faster speeds, young, healthy populations tend to increase the joint torque from the hip, but not the knee or ankle, which is unexpected. Investigating how muscles accelerate an individual during SD could explain the unexpected joint torque pattern, help gain a better understanding of the mechanisms behind SD, and inform rehabilitation. Therefore, the purpose of this study is to examine how individual muscles accelerate the center of mass (COM) during SD in young, healthy subjects at three different speeds. Quantitative gait analysis and dynamic simulations were used to determine muscle forces and their contributions to vertical and horizontal acceleration of the COM. Muscle forces and their contributions to acceleration of the COM generally increased with increasing speed except for during the controlled lowering phase; forces and contributions to vertical acceleration from the soleus, forces from the vastus medialis, and forces and contributions to vertical and horizontal acceleration from the vastus lateralis seemed to decrease with increasing speed. These results will establish a baseline to better evaluate compensatory strategies and deficits in populations that experience difficulty completing SD, and eventually allow for improved rehabilitation for these populations.

Acknowledgements

First, I would like to thank Elena Caruthers. She constantly gave guidance and help in many different aspects of this project, all while being patient as I learned and grew during the process. She went above and beyond to make sure I completed my project on time, even though she had different deadlines than I. I cannot thank her enough for all she taught me and did to help this project. Secondly, I would like to thank Dr. Robert Siston. He initially encouraged me to pursue undergraduate research. I have grown and learned so many valuable life skills from this process and am very happy he took the time to introduce me to research. I would also like to thank him for always helping when needed and encouraging me through the project. Additionally, a big thank you to all the members of the NMBL Lab who were always willing to speak up when I was I need of help. They gave me plenty of constructive criticism, tips, and assistance that I would have been lost without. Finally, I would like to thank my friends and family. They are my support system and encouraged me to keep going when I needed it most.

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Field of Study

Major: Mechanical Engineering

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1 Introduction

Stair climbing (SC), including both stair ascent (SA) and stair decent (SD), is an everyday task but is considered to be a difficult and potentially dangerous activity [1, 2]. An estimated 10% of all adults in America have difficulty climbing stairs [3]. For example, the elderly and those with lower limb disabilities, such as knee osteoarthritis (OA) and those with joint replacements, regularly have more difficulty climbing stairs than young, healthy populations [3]. In order to maintain independence in society, the ability to navigate stairs safely is important for populations in which SC is difficult. Because stair climbing is considered to be a physically demanding, difficult task, especially in comparison to walking, clinicians use a timed stair climbing test to assess patient lower limb function [4]. The stair climbing test times how long it takes an individual to climb up and down the stairs and is considered a good measure of lower limb performance.

Stair descent is considered to be more challenging than SA as four out of five falls on stairs occur during SD [1]. Zachazewski et al. found that SD has a larger separation between the center of mass (COM) and the center of pressure than SA, indicating SD has a greater inherent instability [2]. Further research has been done to investigate the difficulty of SD using experimental methods, such as motion capture and electromyography (EMG), to assess factors such as the kinematics, kinetics, [3-9] and muscle activations [4, 7, 9-11] in healthy and pathological populations. In a young, healthy population, the knee flexion moments [5, 8, 9], knee flexion angles [5, 9], and ankle dorsiflexion angles [5, 8, 9] are greater during SD when compared to those during SA. The quadriceps, gluteal muscles, and plantar flexors have also been found to be less active during SD than SA [9, 11].

Rehabilitation often focuses on these muscles that are used in SD. For example, quadriceps strengthening is a common exercise therapy used to reduce pain and improve performance for patients, but for unknown reasons, quadriceps strengthening and similar techniques leave room for further improvement for patients [12]. For instance, up to 40% of patients diagnosed with knee OA do not have significant improvement in short-term pain or ability to perform activities of daily living like SD after rehabilitation [12]. Furthermore, for those patients who do have initial improvement from rehabilitation, physical function slowly declines and pain increases over time [13]. The reasons for these outcomes are not well understood, therefore, more research is needed to gain a better understanding of the mechanisms behind SD in order to further improve rehabilitation strategies.

Furthermore, since young, healthy populations often perform SD at a faster speed than those with lower limb disabilities, previous studies have also looked at how kinematics, kinetics, and muscle activations change with speed to understand what allows healthy populations to descend stairs at a faster speed than older or pathological populations [4, 9]. In particular, Lewis et al. found that when SD is performed at slower speeds, compared to a self-selected speed, joint torques and muscle activations are reduced at the hip, knee, and ankle [4, 9]. This indicates that slowing down while descending stairs could make the task easier by reducing demand, particularly on the hip flexors, knee extensors, and ankle plantar flexors. When SD is performed at faster speeds, compared to a self-selected speed, young, healthy individuals tend to increase hip joint torques, but not the knee or ankle joint torques [9]. However, during level walking, it was found that joint torques at the hip, knee, and ankle generally increased with increasing speed [14]. Even though walking and SD have different kinematics, they are both over-ground locomotive tasks, so we would expect that they would have similar joint torque patterns with increasing speed. Therefore, more information about what causes these lower limb joint torque patterns is needed to better understand how young, healthy populations descend stairs.

In order to further understand these unexpected joint torque patterns during SD, individual muscle forces needs to be explored. Furthermore, determining individual muscle forces and how those muscles contribute to acceleration of the COM may give clinicians information to specifically target certain muscles that could help improve a patient's functional performance during SD. However, due to the complex dynamics of the human body, experimental methods such as motion capture and electromyography do not have the capability to determine individual muscle behavior, such as muscle forces and how those muscle forces contribute to the acceleration of the COM during SD [15]. In addition, due to a muscle's ability to accelerate joints it does not span and body segments to which it does not attach, a muscle's contributions to acceleration of the COM may be counterintuitive to what is currently known about a muscle's anatomical function and the kinematics of the lower limbs during a task [14, 16-18].

Dynamic simulations, however, can estimate muscle forces and how those muscle forces contribute to the vertical and horizontal acceleration of the COM [15, 19]. Lin et al. used dynamic simulations to investigate muscle contributions to SD at a self-selected speed. This study reported that the quadriceps, plantarflexors (soleus and gastrocnemius), and gluteal muscles were large contributors to vertical acceleration of the COM [11]. In addition, the gluteal muscles and gastrocnemius contributed to horizontal acceleration of the COM while the quadriceps opposed it [11]. While this study investigated muscle contributions to SD during a self-selected speed, a better understanding of how individual muscles contribute to accomplishing SD across speeds is needed, particularly to better understand how muscle contributions influence the increased hip joint torque obtained during SD at a fast speed. In addition, understanding how speed influences the task may help to more accurately classify clinical and statistically significant differences during the stair climbing test [9].

The results of investigating how young, healthy populations descend stairs across a range of speeds can also be used to establish a baseline to better identify compensatory strategies and deficits in older and pathological populations. By first gaining a better understanding of how young, healthy populations complete these tasks, an initial baseline can be established which can identify what muscles this population is using to complete SD. After investigating how young, healthy populations complete this tasks, the next step is to investigate older, healthy populations and compare them to the baseline and then do the same with pathological populations. The order of these studies will allow compensatory strategies and deficits to be identified in older and pathological populations, which could lead to more informed rehabilitation efforts and help further improve functional performance for those populations during tasks like SD. Additionally, doing comparable studies on each of these populations will help to identify differences between these populations, which may help explain what allows to certain populations, like young healthy individuals, to climb stairs faster than other populations.

1.1 Focus of Thesis

The purpose of this project was to examine how individual muscles contribute to SD in a young, healthy population at different speeds. This was done by examining individual muscle forces and their contributions to horizontal and vertical acceleration of the COM (i.e. muscle function) during SD at slow and fast speeds. Next, the muscle forces and their contributions to horizontal and vertical acceleration of the COM during SD at slow and fast speeds were compared to each other and compared to self-selected (SS) speed (Figure 1.1). Based on a previous study which investigated walking at different speeds [14], I hypothesized that as descending speed increases, both muscle forces and contributions to the horizontal and vertical acceleration of the COM from the quadriceps, gluteal muscles, and plantarflexors would increase in a young, healthy population.

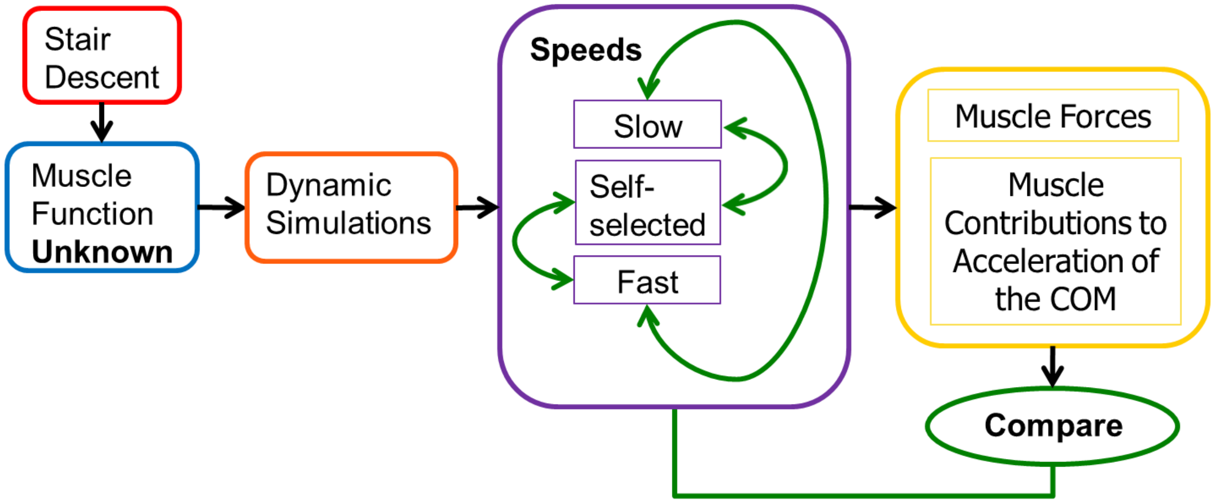

Figure 1.1: Flow Chart of Study's Approach

1.2 Overview of Thesis

This thesis contains 5 chapters. Chapter 1 provides background information about the stair descent task and motivation for creating simulations of a young, healthy population. Chapter 2 discusses the methodology, including data collection, data analysis, dynamic simulations, and statistical analysis. This describes how muscle forces and their contributions to acceleration were calculated and analyzed across muscles, speeds, and phases. Chapter 3 gives the results from the data, consisting of muscle forces and their contributions to horizontal and vertical accelerations of the COM. Chapter 4 discusses the implications from the results obtained and limitations to this work. Chapter 5, the conclusion, summarizes the contributions of this work and proposes future work.

2 Methodology

2.1 Data Collection

The data used in this study was previously collected by a former PhD student, Jackie Lewis in a study approved by the Institutional Review Board at The Ohio State University. Thirty young, healthy subjects (15 males, 15 females, age=27.5±10.7 years, height=1.8±0.08 m, weight=73.9±12.6 kg) were tested under the same quantitative gait analysis protocol [9]. Each subject was asked to climb stairs at three different speeds (slow, SS, fast). Each subject performed the SA and SD trials on a custom-made three-step staircase (tread depth: 25.5 cm, step height: 20 cm) (Figure 2.1). For each speed, at least three trials starting with the left leg and three trials starting with the right leg were collected. The subjects stepped forward with their preferred limb first and were then instructed to step with a specific limb, if necessary, to collect all trials. First, the subjects were told to climb at their normal SS speed. They then were instructed to climb slower than their SS speed (slow) and then faster than their SS speed (fast) without having a flight phase (i.e. running).

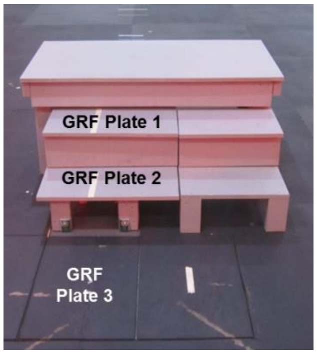

Figure 2.1: Custom-made stair case with locations of GRF plates shown

A modified point-cluster marker set was used on the upper and lower extremities bilaterally during testing (Figure 2.2). The modified point-cluster marker set minimizes skin artifact and establishes a tracking coordinate system to prescribe the motion of the subject during dynamic movement [20]. Ten Vicon MX-F40 cameras (Vicon; Oxford, UK) were used to capture the three-dimensional trajectories of the skin-mounted reflective markers at 150 Hz. Additionally, during each trial, the ground reaction forces (GRFs) and muscle activations were collected. The GRFs were sampled from three force platforms (Bertec, Columbus, OH) at 1500 Hz for the first two steps and the floor in front of the staircase (Figure 2.1). A 16-channel wireless surface EMG (Telemyo DTS, Noraxon USA, Inc., Scottsdale, AZ), sampled at 1500 Hz, was used to collect lower limb muscle activations. The electrodes for EMG were placed according to the SENIAM model [21]. Before placement, electrode locations were shaved, lightly abraded, and cleaned with alcohol pads. Pre-gelled, rectangular Ag/AgCL surface dual electrodes with a 42 mm inter-electrode distance (Vermed, Inc; Bellows Falls, VT) were placed unilaterally on the following muscles of a randomly assigned limb: gluteus maximus, gluteus medius, rectus femoris, vastus lateralis, vastus medialis, semimembranosus, biceps femoris (long head and short head), tibialis anterior, medial gastrocnemius, lateral gastrocnemius, and soleus (Figure 2.3).

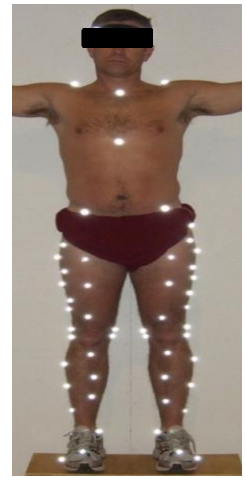

Figure 2.2:Modified point-cluster marker set used during motion capture

2.2 Data Analysis

From the collected data, one SD trial per subject was selected to be further analyzed with dynamic simulations for each speed (slow, SS, and fast) based on marker visibility and quality of the raw EMG. Some of the subject data was excluded from further analysis based on the marker visibility and if the subject had previous lower extremity injuries, lower extremity surgery, or open abdominal surgery. We included 8 subjects in our analysis (3 males, 5 females; age: 22±1.5 years; weight: 75±14 kg; height: 1.77±0.07m).

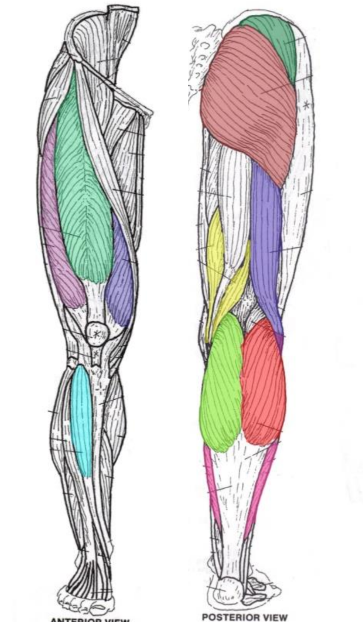

Figure 2.3: EMG muscles. EMG was taken on each of the colored muscles

2.3 Dynamic Simulations

OpenSim 3.1, an open-source software system that is widely used in biomechanics applications, was used to create dynamic simulations of each subject's slow, SS, and fast SD trial [15]. The generic musculoskeletal model, the Full Body Model 2015 (Figure 2.4), used for these simulations was created by my research mentor, Elena Caruthers [22]. The Full Body Model was chosen since it has the ability to capture possible lower back and arm movement during SD. Five steps were completed to analyze the data: scaling, inverse kinematics, residual reduction algorithm, static optimization, and induced acceleration analysis [15]. From these steps, the subject's muscle forces and muscle contributions to horizontal and vertical acceleration of the COM were estimated.

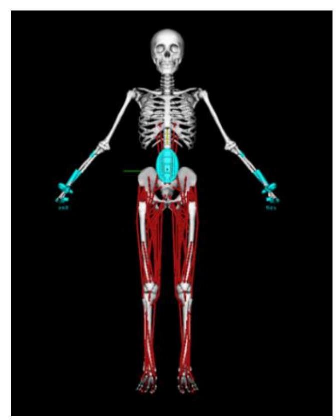

Figure 2.4: Full Body Model 2015

In step 1, the generic musculoskeletal model is scaled so that it reflects the mass distributions and dimensions of each body segment for each subject [15]. The dimensions of each body segment in the model are scaled based on relative distances between pairs of markers obtained from motion capture during the static calibration trial and the corresponding virtual marker locations in the model [15]. The body segment mass properties are scaled proportionally so the total mass of the model is equal to the total measured mass of the subject [15]. This step was completed by my research mentor. The remaining four steps were completed by me for the slow and fast SD trials, while my research mentor completed the four steps for the SS SD trials.

In step 2, inverse kinematics (IK) determines the model's joint angles and translations that best reproduce the raw marker data experimentally collected from motion capture [15]. This is framed as a least-squares problem to minimize the difference between the experimental marker locations from motion capture and the model's virtual marker locations [15]. During IK, each marker is assigned a weight to specify how heavily the virtual marker should track the experimental marker, where a marker with a higher weight is tracked more heavily. The thigh and shank cluster reference frames, established from point-cluster technique, and the bony landmarks of the pelvis and feet were weighted most heavily. Inverse dynamics (ID) then uses the motion from IK to compute the model's joint torques about the hip, knee, and ankle [15].

After IK is performed, the model may not be dynamically consistent with the experimental GRF and accelerations due to effects of modeling and processing the data

[15]. In Step 3, residual reduction algorithm (RRA) is applied to minimize these errors so that the motion satisfies Newton's Second Law (force=mass*acceleration) and is dynamically consistent with the experimental GRFs. To obtain consistency, RRA calculates three residual forces and three residuals moments (one force and moment for each direction) that are applied to the model's pelvis. It is required that the residuals forces and moments are made as small as possible while maintaining dynamic consistency [15]. To minimize these residual values, RRA alters the placement of the torso COM of the subject-specific model, varies the kinematics from step 2 (IK) to make them consistent with the experimental GRFs, and slightly changes the model's body segment mass properties [15]. The residual forces were acceptable if the value was below 25 N and the residual joint torques were acceptable if the value was below 75 Nm. RRA was run multiple times to the point at which the model's total recommended mass change was less than 0.05 kg.

In Step 4, static optimization (SO) resolves net joint torques into individual muscle forces by using an objective function to resolve the issue of muscle redundancy [15, 23]. For this study, we chose to use the objective function of minimizing the sum of squared muscle activation, due to its association with minimized energy expenditure, which humans are believed to desire to achieve during movement [24]. The SO simulated activations for the muscles with experimental EMG were compared after normalizing the EMG by the peak value of each muscle's simulated activation [25], which has been done in previous studies [26, 27]. Upper and lower bounds were applied to the simulated activations so that they would more closely mimic the experimental

EMG. Multiple iterations of applying bounds were performed to allow for better agreement of the SO activations and the experimental EMG (Figures 2.5, 6, 7). The SO forces were verified that they were from muscle-tendon actuators and not from reserve actuators by comparing the hip, knee, and ankle normalized RRA joint torques (%BW*ht) to those calculated from SO (sum of the product of muscle forces and the corresponding moment arms) (Figures 2.8, 9, 10).

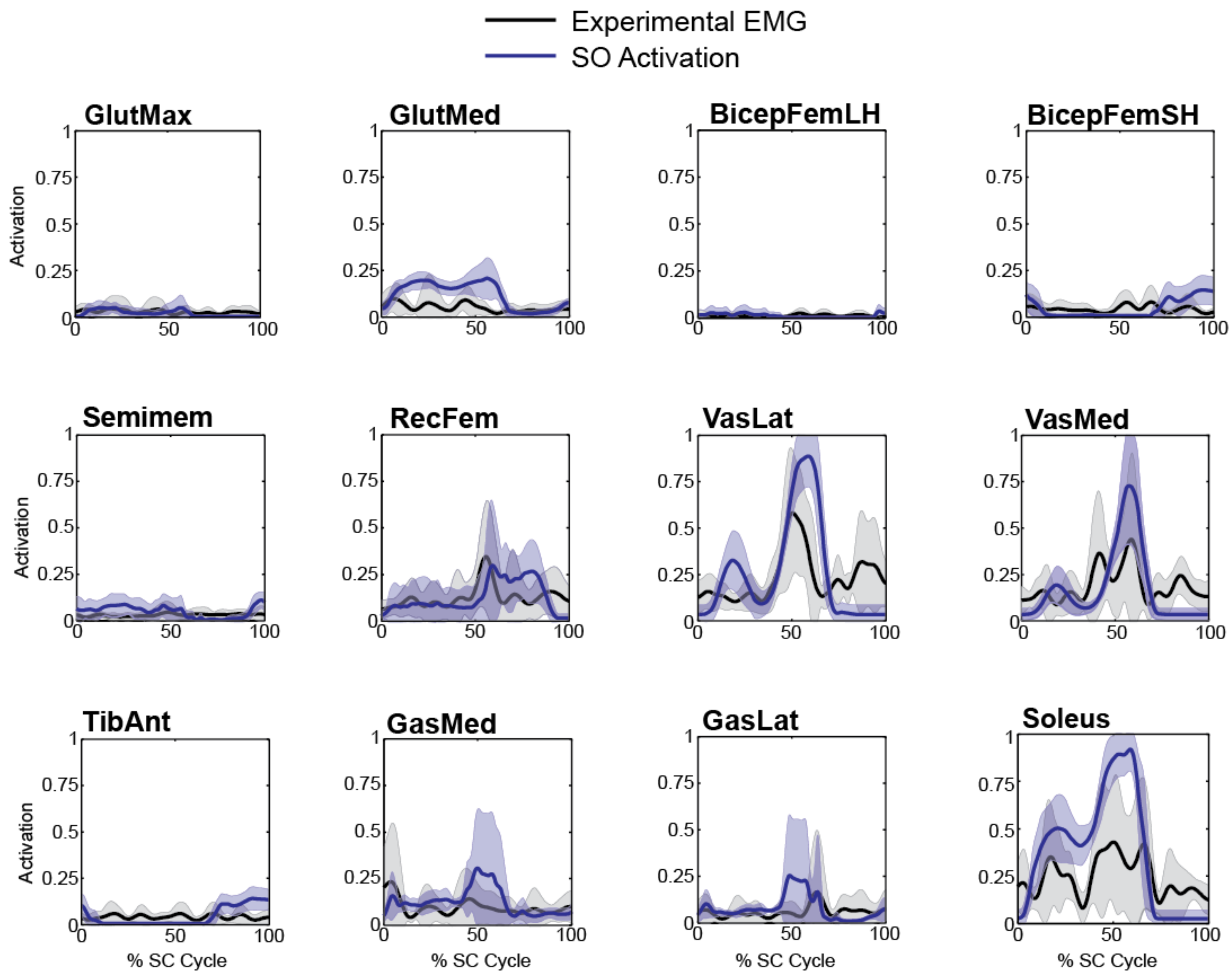

Figure 2.5: Average SO activations (blue) and experimental EMG (black) for one stair climbing (SC) cycle performed at a slow speed. The shaded regions represent one standard deviation. The peak value of the experimental EMG is normalized to the peak value of the simulated muscle activation. A reading of "1" indicates 100% activation.

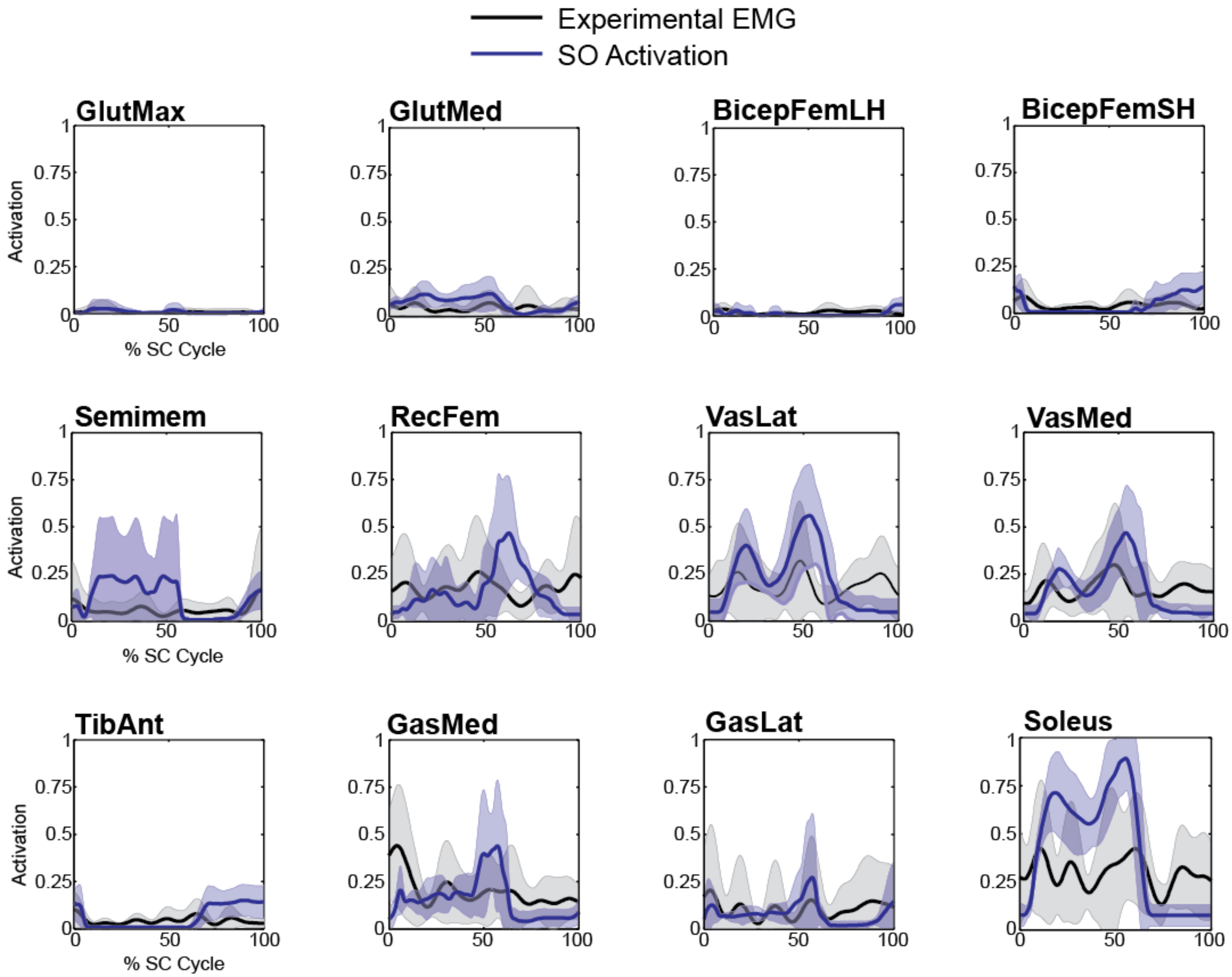

Figure 2.6: Average SO activations (blue) and experimental EMG (black) for one stair climbing (SC) cycle performed at a SS speed. The shaded regions represent one standard deviation. The peak value of the experimental EMG is normalized to the peak value of the simulated muscle activation. A reading of "1" indicates 100% activation.

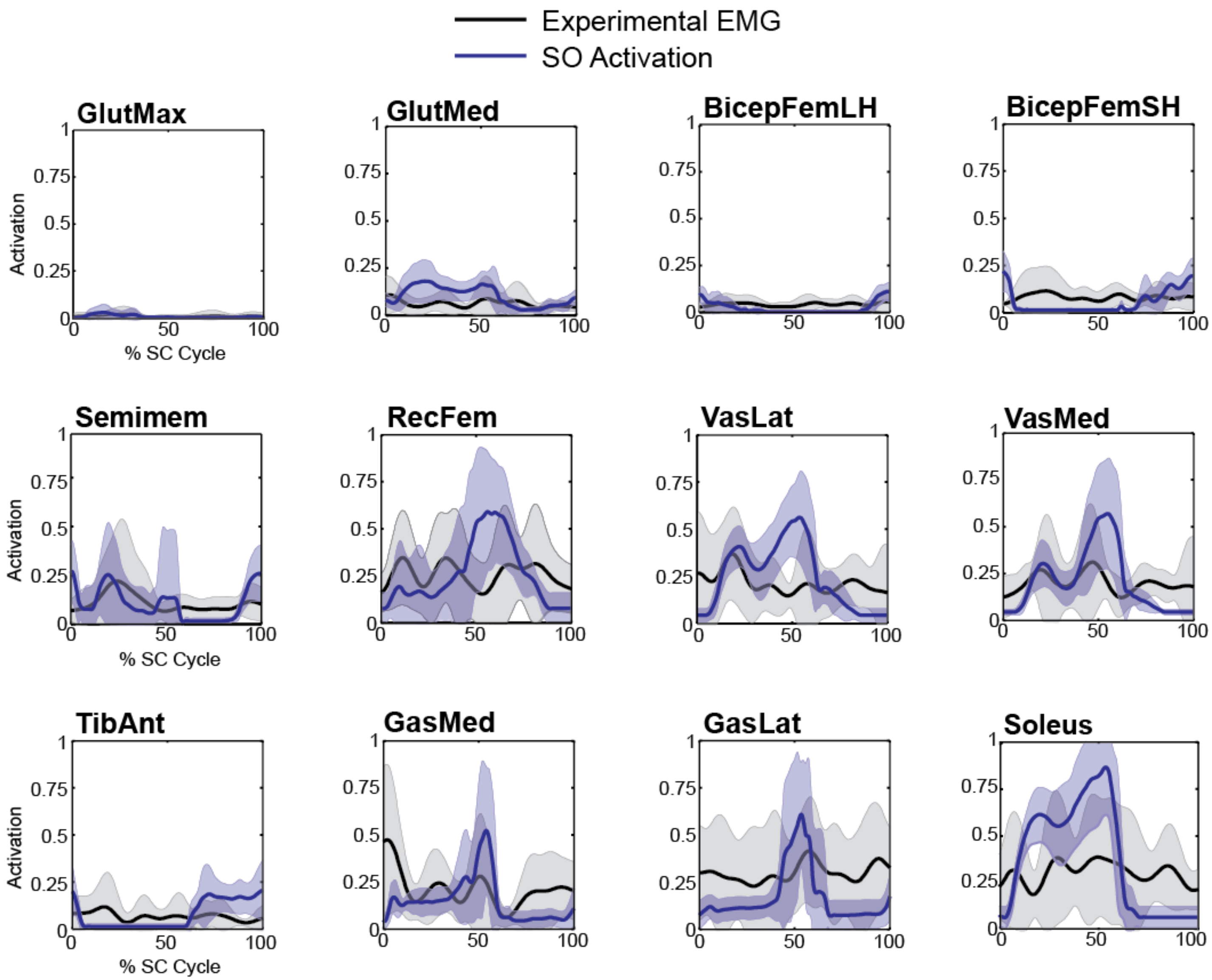

Figure 2.7; Average SO activations (blue) and experimental EMG (black) for one stair climbing (SC) cycle performed at a fast speed. The shaded regions represent one standard deviation. The peak value of the experimental EMG is normalized to the peak value of the simulated muscle activation. A reading of "1" indicates 100% activation.

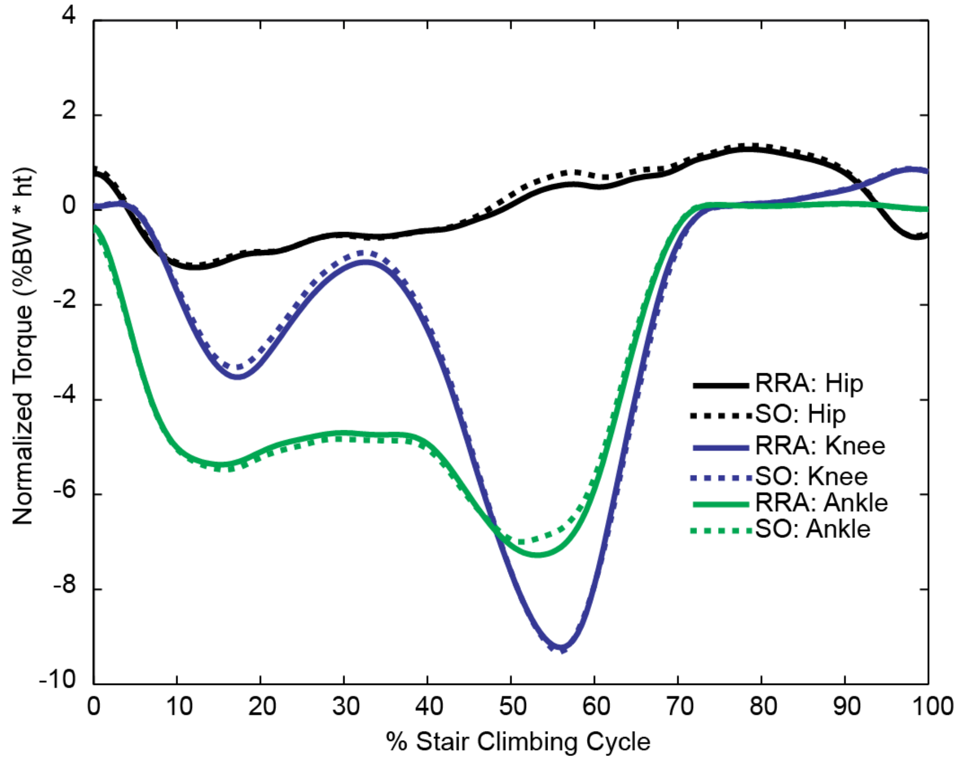

Figure 2.9: Comparison of average joint torques from RRA and from SO (muscle force multiplied by moment arm) for the hip, knee, and ankle for one stair climbing cycle performed at a slow speed. All torques are external.

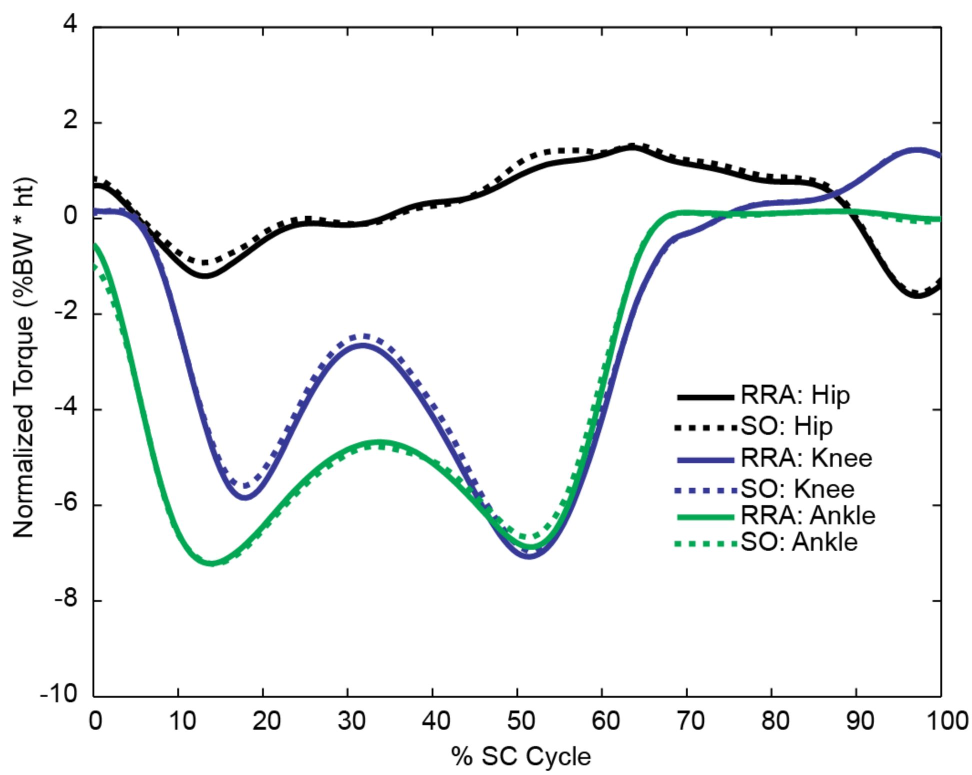

Figure 2.8: Comparison of average joint torques from RRA and from SO (muscle force multiplied by moment arm) for the hip, knee, and ankle for one stair climbing cycle performed at a SS speed. All torques are external.

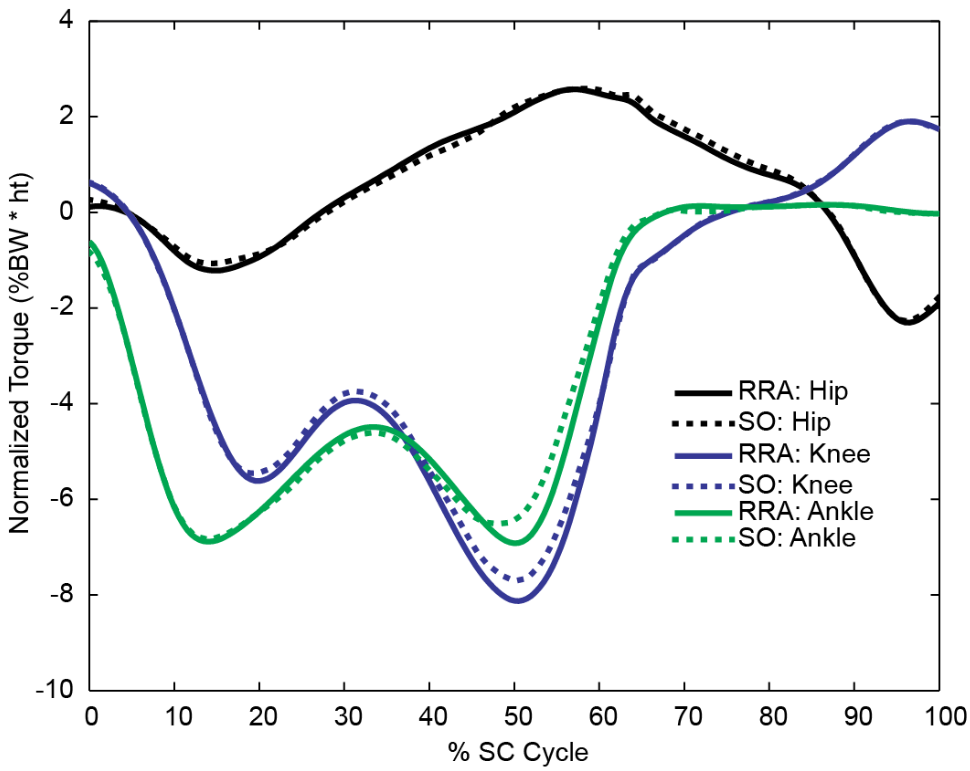

Figure 2.10: Comparison of average joint torques from RRA and from SO (muscle force multiplied by moment arm) for the hip, knee, and ankle for one stair climbing cycle performed at a fast speed. All torques are external.

In Step 5, induced acceleration analysis (IAA) is used to determine how the calculated muscle forces are contributing to the horizontal and vertical acceleration of the model's COM at each time step of the simulation [17]. IAA can compute the potential of each muscle to accelerate the COM for each time step. The product of the muscle force, calculated from SO, and the potential of the muscle for each time step, equals each muscle's contribution to the horizontal and vertical acceleration of the model's COM across the entire motion. The

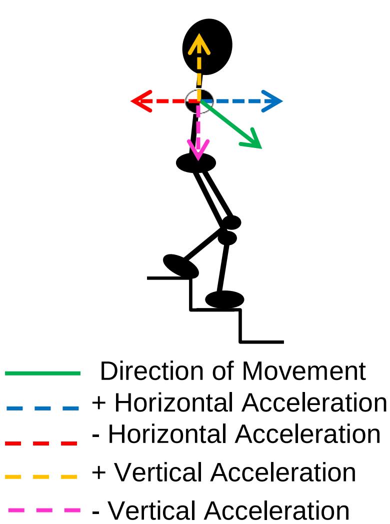

Figure 2.11: Convention used to define acceleration directions

horizontal acceleration will be defined as the acceleration component parallel to the top of a stair step whereas vertical acceleration will be defined as the acceleration component orthogonal to the top of a stair step (Figure 2.11). The remaining contributions to the acceleration of the COM from skeletal alignment and velocity effects (i.e. centripetal and Coriolis forces) were estimated by subtracting the total muscle-induced accelerations from the accelerations due to GRFs (Figures 2.12, 13, 14) [14, 28].

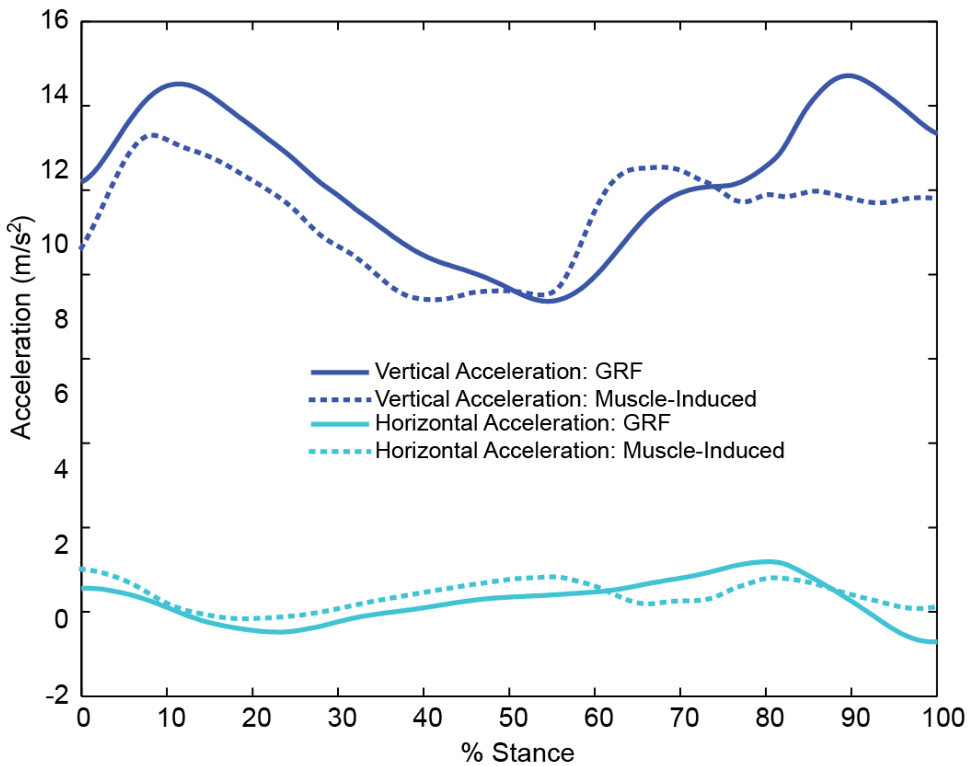

Figure 2.12: Total average from contributions to the vertical and horizontal acceleration of the COM from ground reaction forces (GRFs) and those induced by muscles across the stance phase of the stair climbing cycle performed at a slow speed.

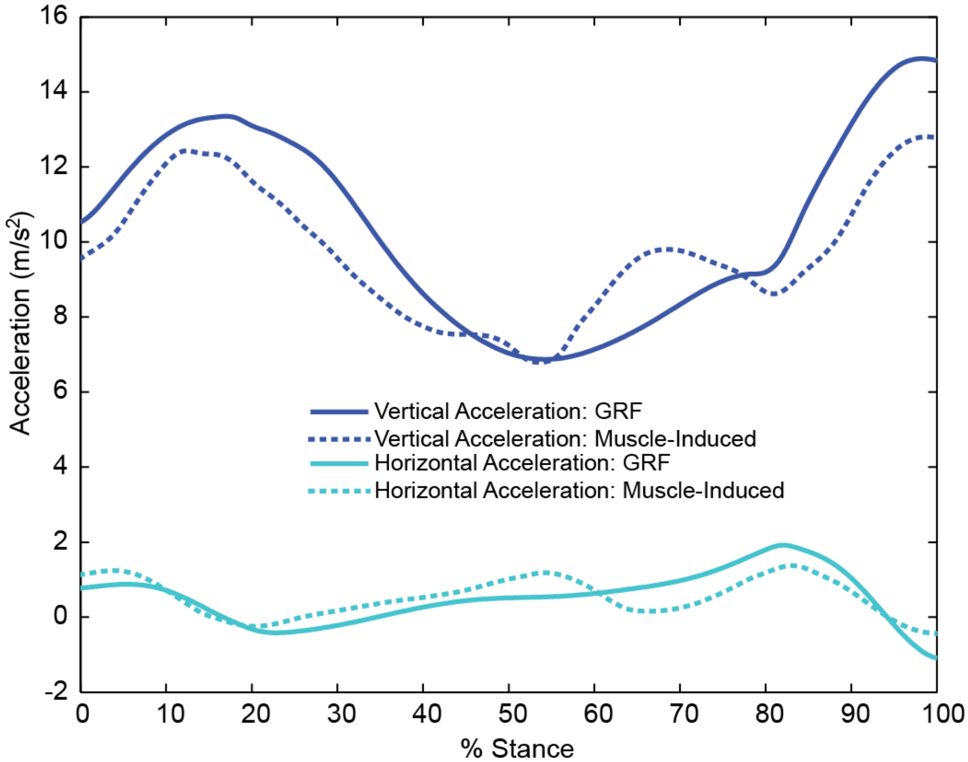

Figure 2.14: Total average contributions to the vertical and horizontal acceleration of the COM from ground reaction forces (GRFs) and those induced by muscles across the stance phase of the stair climbing cycle performed at a SS speed.

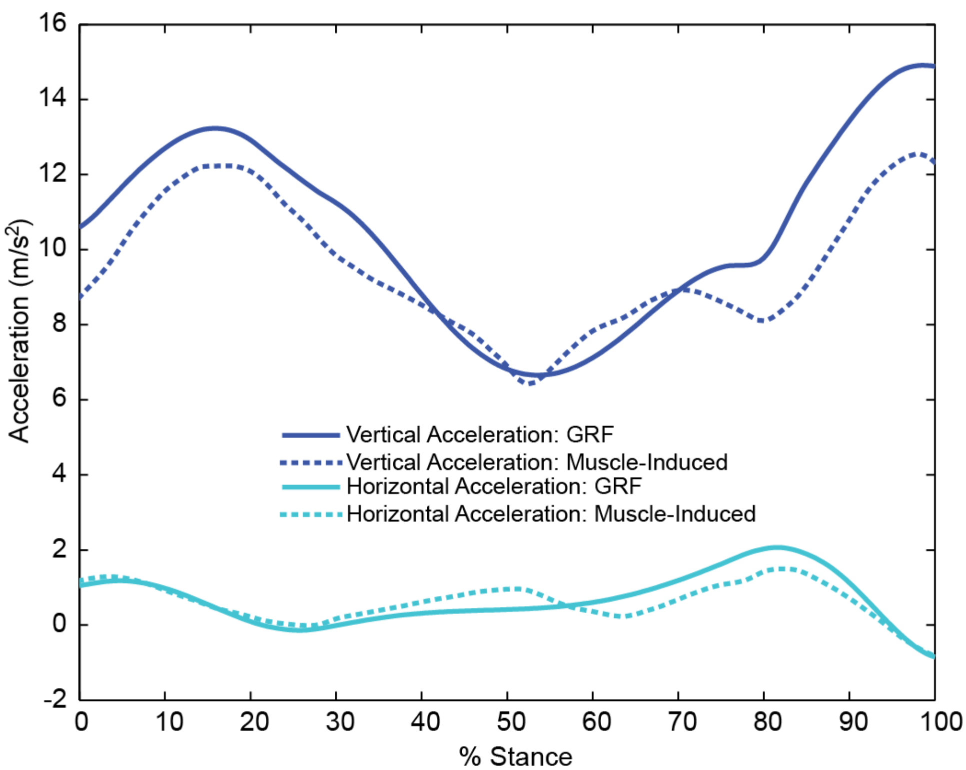

Figure 2.13: Total average contributions to the vertical and horizontal acceleration of the COM from ground reaction forces (GRFs) and from the sum of those induced by muscles across the stance phase of the stair climbing cycle performed at a fast speed.

2.4 Analysis

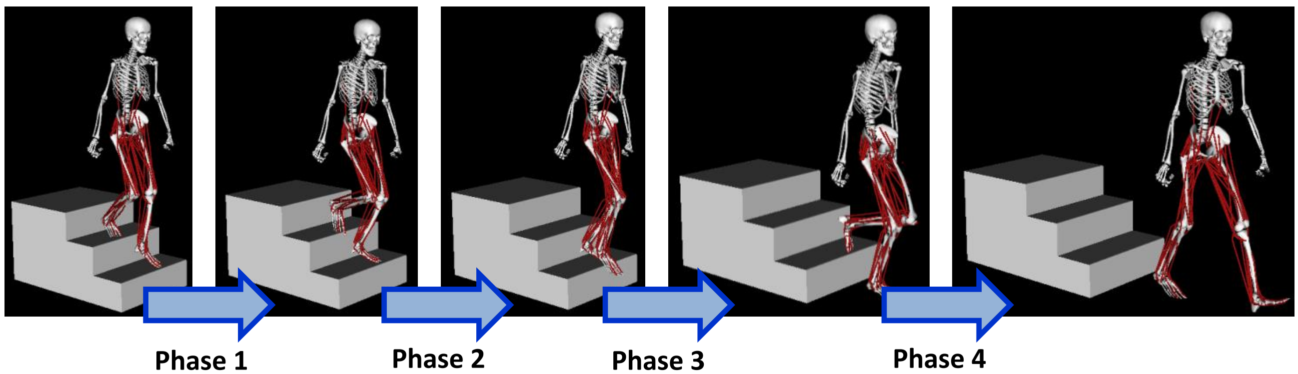

Figure 2.15: Phases 1, 2, 3, and 4 during the SD cycle

The SD cycle was divided into 4 phases: weight acceptance, forward continuance, controlled lowering, and swing [2] (Figure 2.15). During Phase 1 (weight acceptance), the stance limb is loaded. Phase 1 starts when the stance limb strikes stair 2 and ends when the contralateral limb leaves stair 1. During Phase 2 (forward continuance), the body moves forward while being supported by the single limb in stance. Phase 2 starts when the contralateral limb (non-stance limb) leaves stair 1 and ends at mid-stance during single limb support. During Phase 3 (controlled lowering), the whole body COM is lowered. Phase 3 starts at mid-stance during single limb support and ends when the stance limb leaves stair 2. During Phase 4 (swing), the stance limb becomes the swing limb and the body moves forward with the swing limb moving toward foot placement. Phase 4 starts when the stance limb leaves stair 2 and ends when the stance limb strikes the floor. Stance includes phases 1, 2, and 3.

We averaged muscle forces and their contributions to the acceleration of the COM across subjects for each speed in each phase of the SD cycle and summarized them with descriptive statistics (mean and standard deviation). Since the gluteus maximus and medius were modeled as multiple actuators in the Full Body Model, their forces and induced accelerations were calculated by summing the values for each actuator. Separate three-way repeated measures analyses of variance (ANOVA) were used to inspect average maximum muscle forces and induced accelerations across each phase of SD for each speed. Effects investigated were muscle, speed, phase and the interactions between them. Tukey post-hoc pairwise comparisons were used to investigate muscle forces and contributions to the acceleration of the COM as appropriate. Statistical tests were performed in Minitab® Statistical Software (Minitab Inc, State College, PA), and the level of significance was $\alpha = .05$.

3 Results

3.1 Joint Kinematics

The peak hip flexion, knee flexion, and ankle plantarflexion joint angles do not seem to increase with increasing speed (Figure 3.4). Peak magnitudes and patterns for the hip, knee, and ankle are consistent with Lewis et al. (2015) [9].

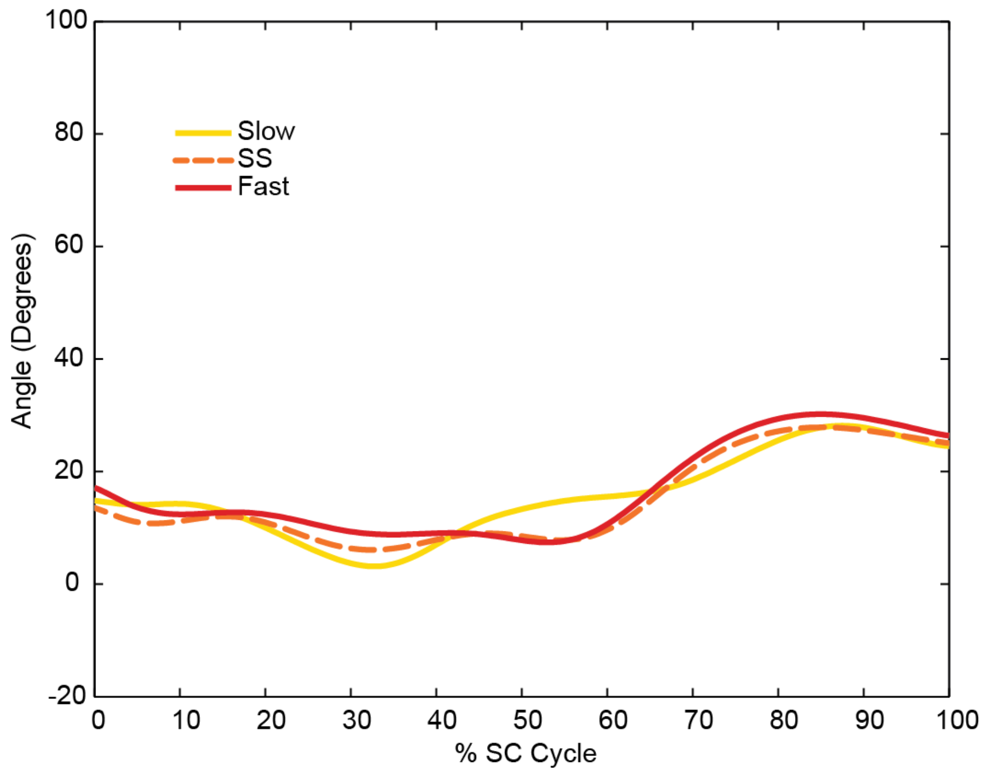

Figure 3.1: Average hip joint angles for each speed across one stair climbing cycle.

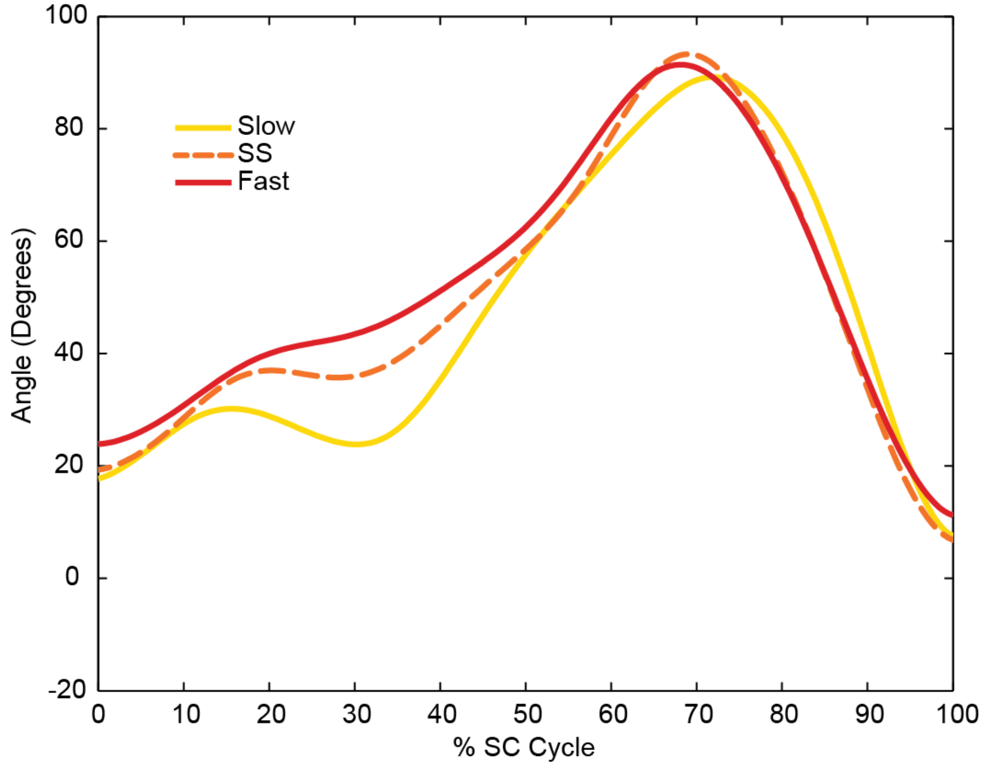

Figure 3.2: Average knee joint angles for each speed across one stair climbing cycle.

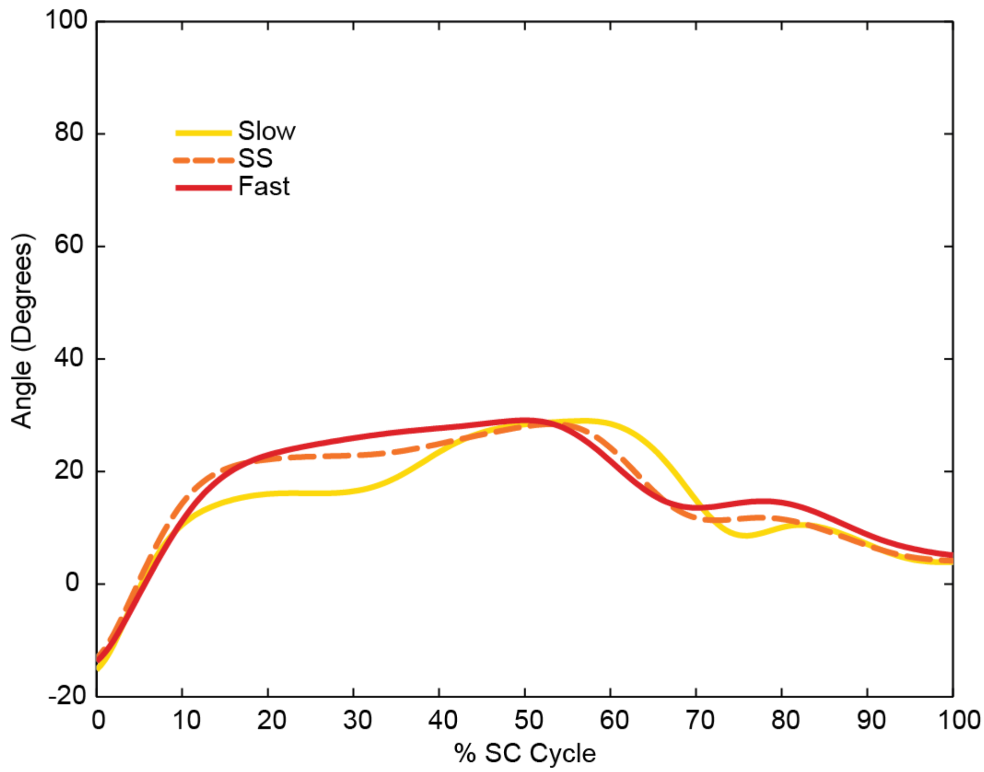

Figure 3.3: Average ankle joint angles for each speed across one stair climbing cycle.

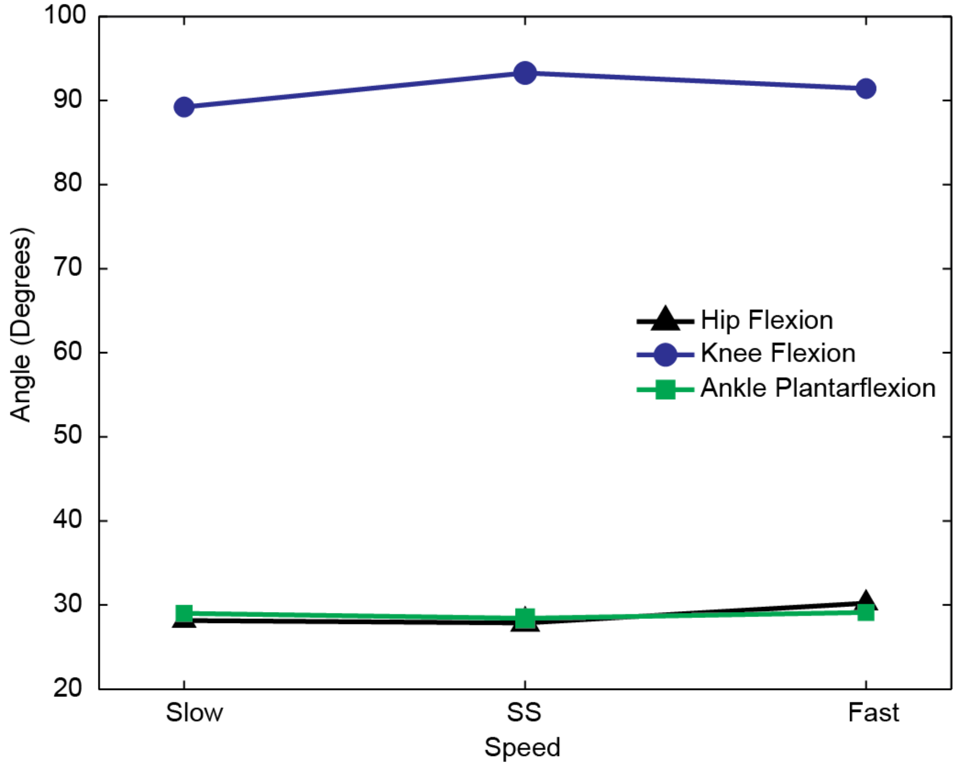

Figure 3.4: Average maximum joint angles for the hip, knee, and ankle for each speed.

3.2 Joint Kinetics

The peak hip extension torques seem to increase with increasing speed, while the peak knee flexion and ankle plantar flexion torques do not (Figure 3.8). This is consistent with the joint torque patterns found by Lewis et al. (2015) [9].

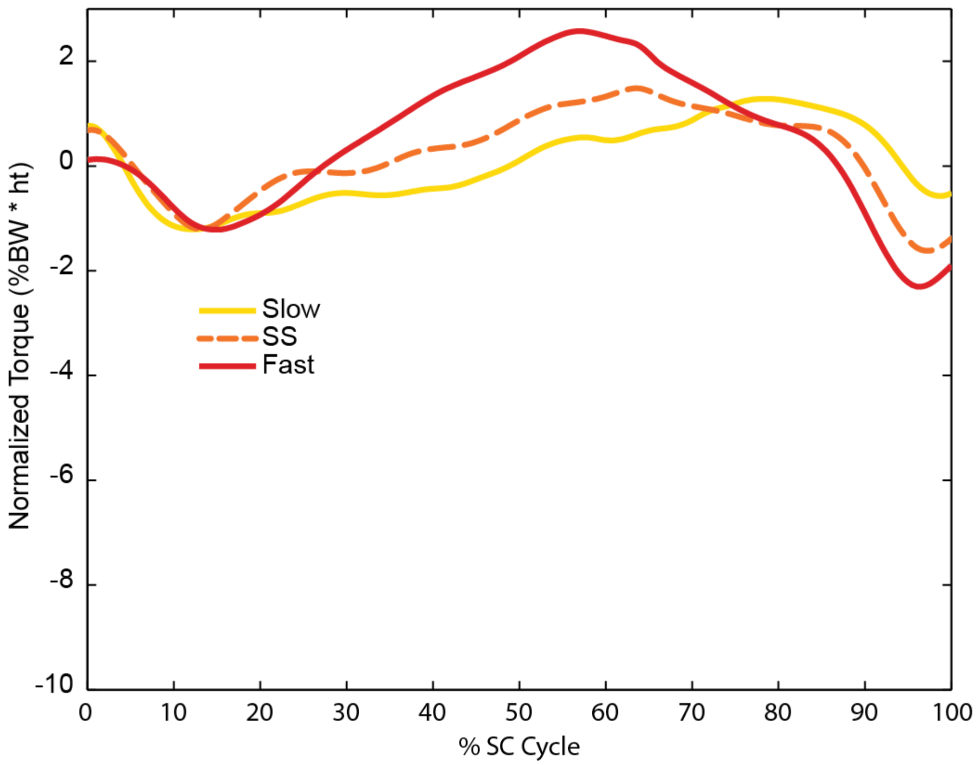

Figure 3.6: Average hip joint torques for each speed across one stair climbing cycle.

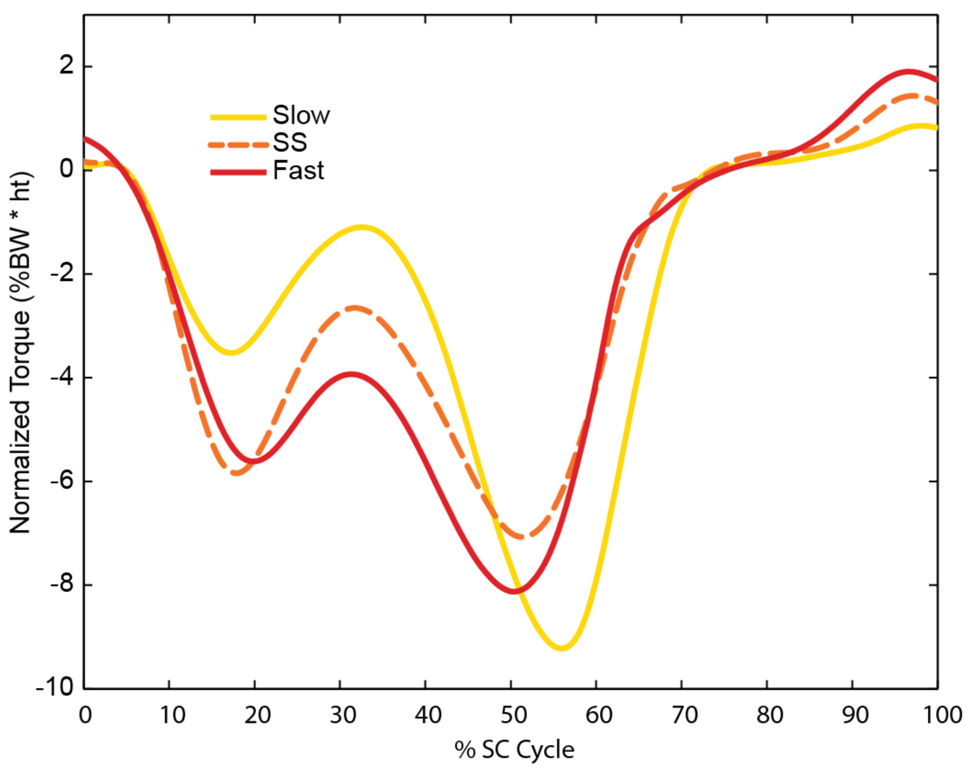

Figure 3.5: Average knee joint torques for each speed across one stair climbing cycle.

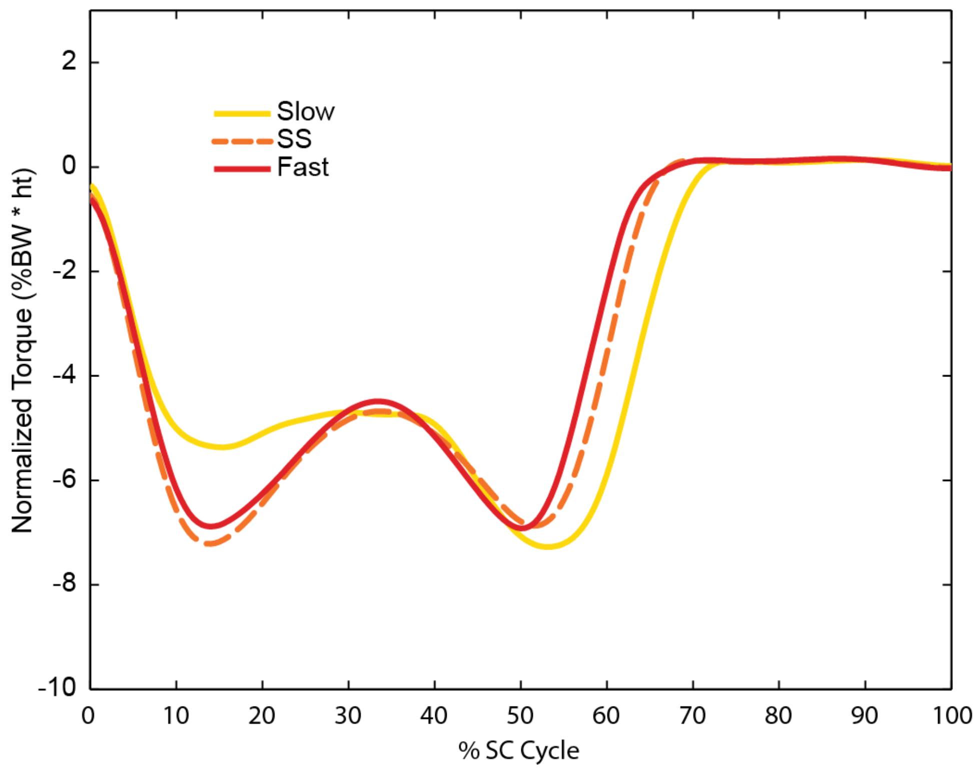

Figure 3.7: Average ankle joint torques for each speed across one stair climbing cycle.

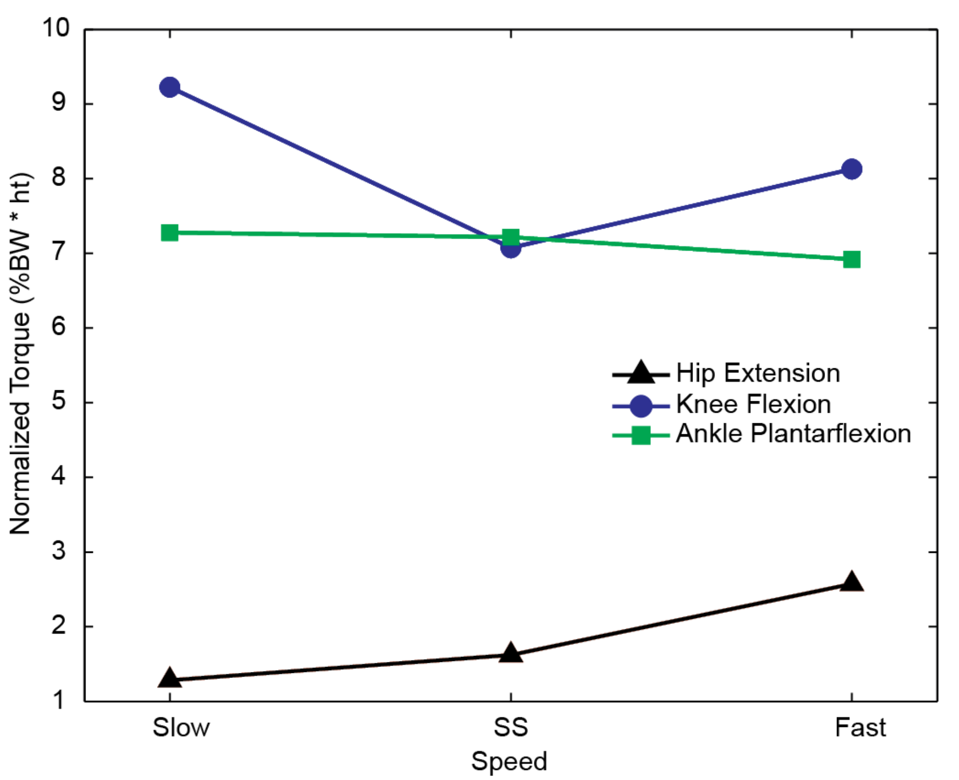

Figure 3.8: Average maximum joint torques for the hip, knee, and ankle for each speed across one stair climbing cycle.

3.3 Muscle Forces

There were significant differences in muscle forces produced between muscles ($p<0.001$), phases ($p<0.001$), and speeds ($p<0.006$). There were also significant interactions between muscle and phase ($p<0.001$), muscle and speed ($p<0.004$), speed and phase ($p<0.001$), and muscle, phase, and speed ($p<0.008$). These outcomes indicate that the muscles produced different forces from one another and across different phases and speeds of SD. The soleus (Sol) produced the largest force across phases 1, 2, 3, and 4 for all speeds (1544.83±884.87 N, $p<0.001$) (Table 3.1). The vastus lateralis (VasLat) produced the next largest force across phases 1, 2, and 3 for each speed (907.44±643.94 N, $p<0.001$) (Table 3.1). Across speeds, the VasLat produced a significantly greater force at slow speed than at SS or fast speed during phase 3 ($p=0.0007$) (Figure 3.13, Table 3.3). The Sol also produced a significantly greater force at slow speed than at SS speed in Phase 2 ($p=0.0007$) (Figure 3.14, Table 3.2). Average maximum muscle forces for the iliacus (Iliac), gluteus medius (GlutMed), gluteus minimus (GlutMin), semimembranosus (Semimem), rectus femoris (RecFem), vastus intermedialis (VasInt), vastus medialis (VasMed), medial gastrocnemius (GasMed), peroneus longus (PerLong), and tibialis posterior (TibPost) are also reported as these muscles produced a force greater than 300 N in one or more phases (Figures 3.12, 13, 14 and Tables 3.2, 3, 4, 5). The remaining lower limb muscles produced forces less than 300 N and are not reported, including the tibialis anterior, gluteus maximus, and lateral gastrocnemius that EMG was reported for. There were more significant differences found between these muscles within the same speed and phase and between phases for a certain muscle at a certain speed, but these are not displayed as our analysis in only concerned about changes within a phase across speeds.

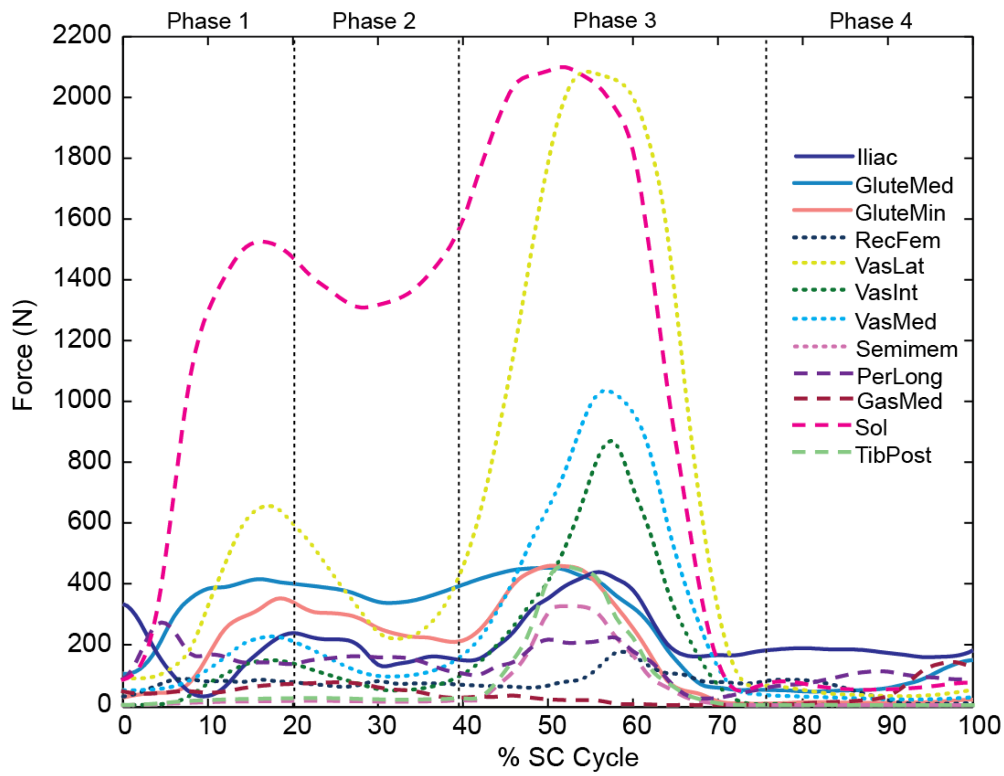

Figure 3.9: Waveform of average muscle forces across one stair climbing cycle (SC) performed at a slow speed.

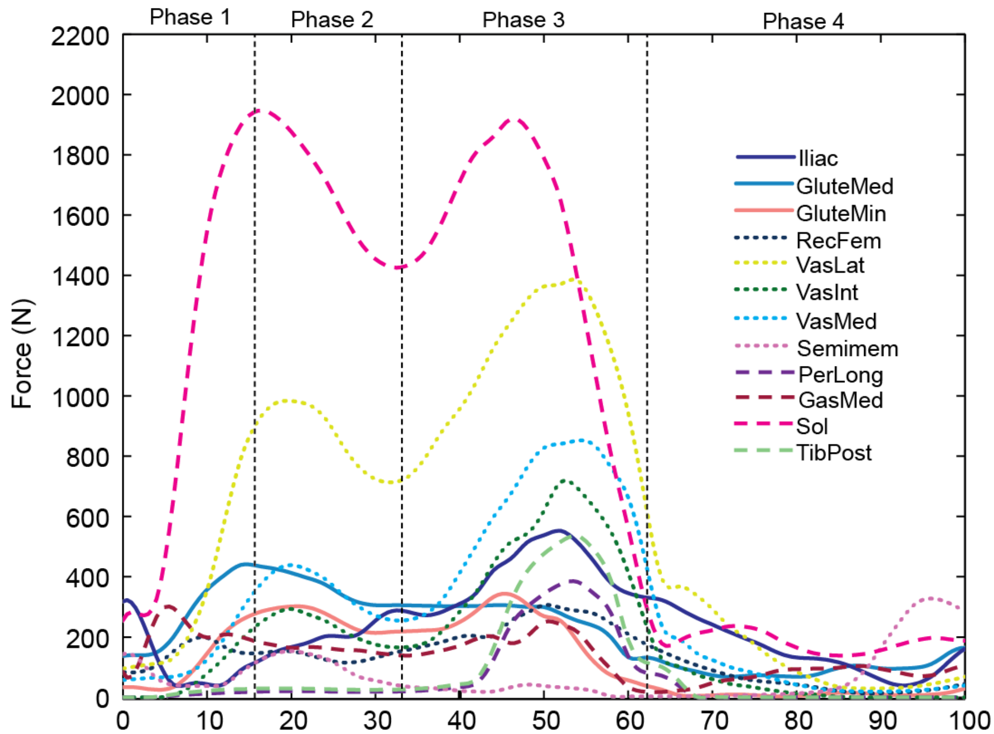

Figure 3.10: Waveform of average muscle forces across one stair climbing cycle (SC) performed at a fast speed.

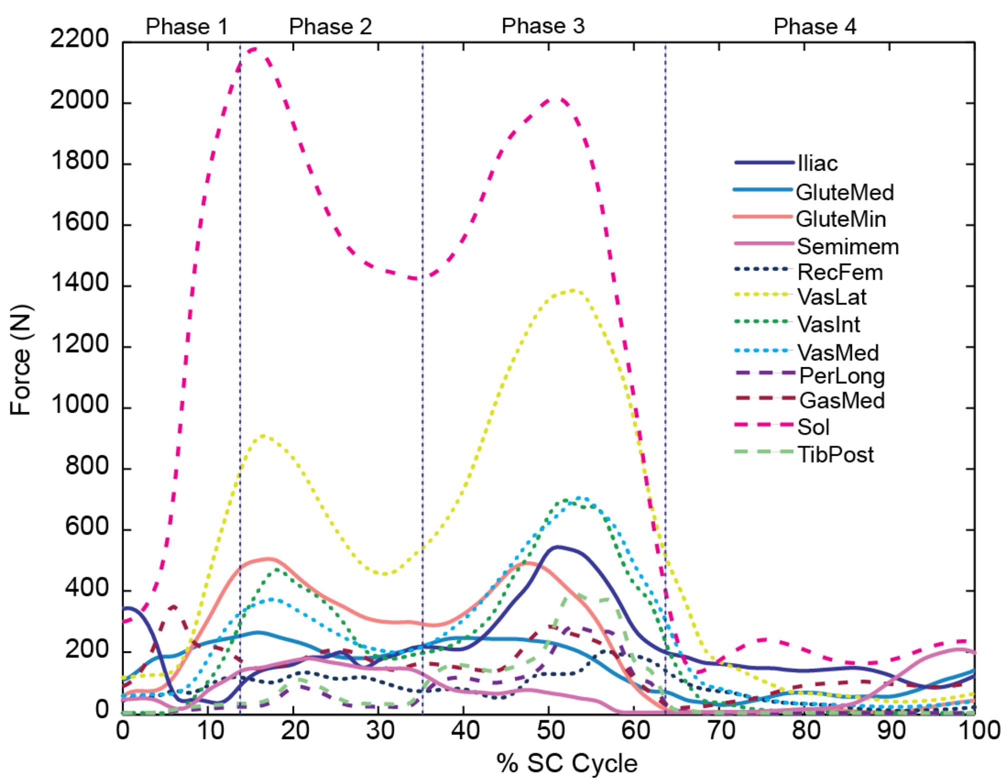

Figure 3.11: Waveform of average muscle forces across one stair climbing cycle (SC) performed at a SS speed.

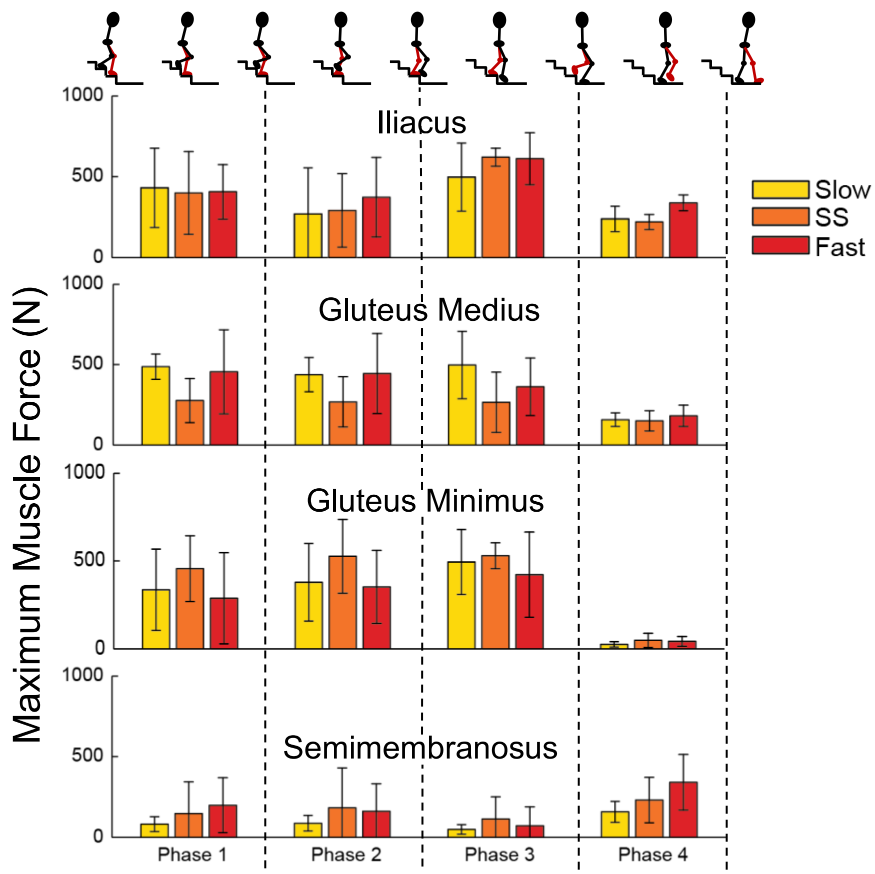

Figure 3.12: Average maximum muscle forces for the iliacus, gluteus medius, gluteus minimus, and semimembranosus muscles for each speed within each phase. Error bars span ± one standard deviation.

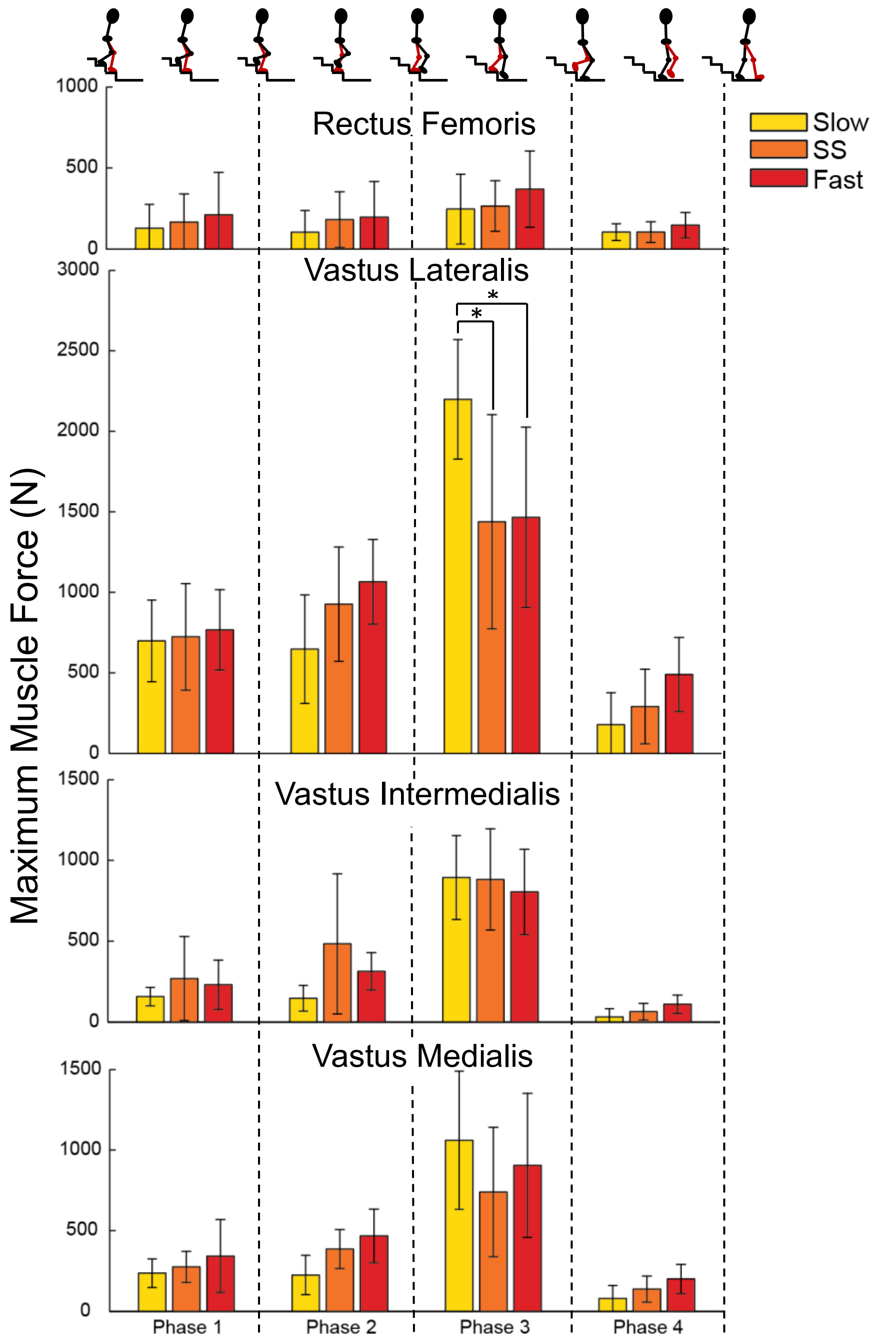

Figure 3.13: Average maximum muscle forces for the quadriceps for each speed within each phase. Error bars span ± one standard deviation. An asterisk (*) indicates that the force generated by the muscle was significantly different between the respective speeds within the phase (p=0.0007).

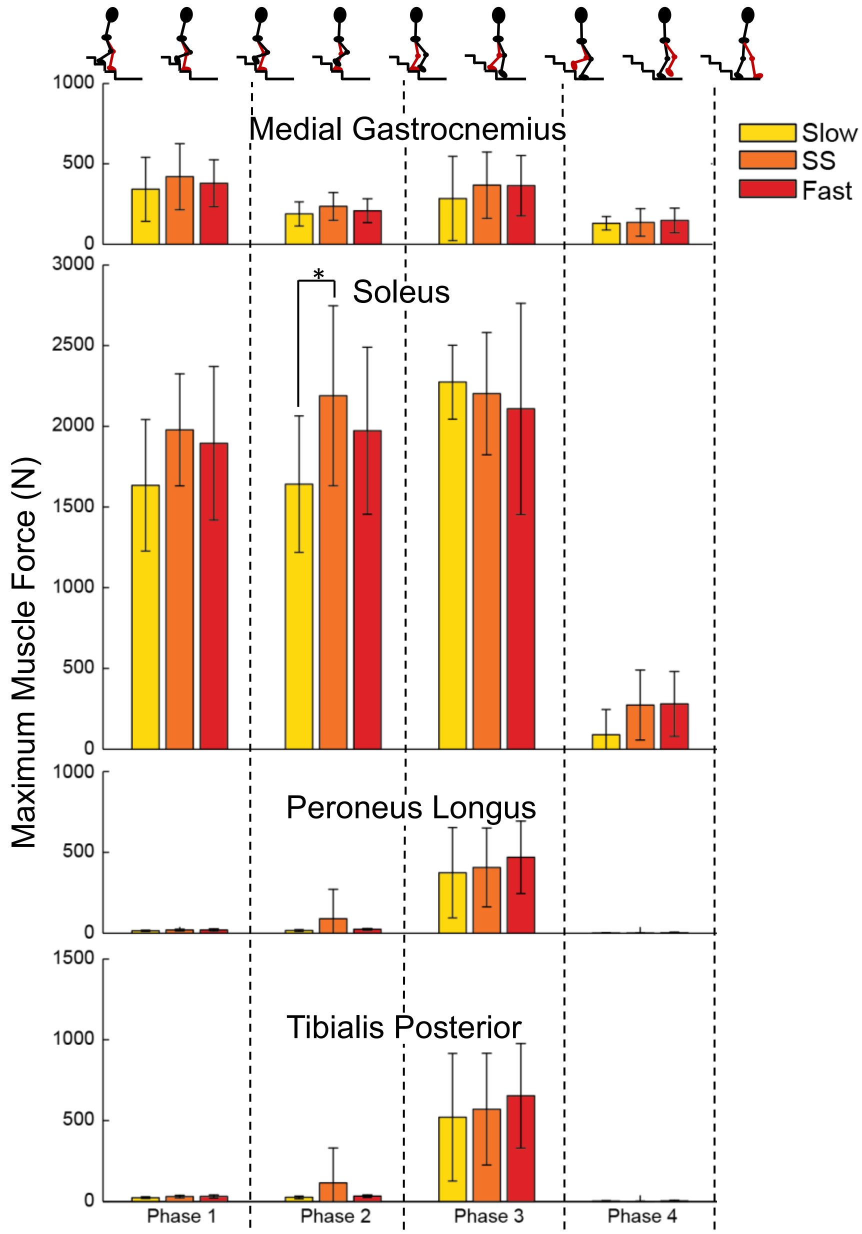

Figure 3.14: Average maximum muscle forces for posterior muscles of the leg for each speed within each phase. Error bars span ± one standard deviation. An asterisk (*) indicates that the force generated by the muscle was significantly different between the respective speeds within the phase (p=0.0007).

Table 3.1 :Mean muscle force across all speeds and all phases. The values represent the mean and standard deviation of all forces produced by a muscle. Means that do not share a letter are significantly different (p<0.05).

Muscle	Mean Muscle Force (N)	Grouping
Sol	1544.83±884.87	A
VasLat	907.44±643.94	B
VasMed	421.26±378.46	C
Iliac	391.80±291.66	C
VasInt	365.63±369.49	C D
GlutMed	332.11±197.47	C D
GlutMin	325.33±325.33	C D
GasMed	267.07±176.46	D E
RecFem	185.75±178.96	E F
TibPost	167.54±301.34	E F
Semimem	151.85±156.78	F
PerLong	119.90±216.47	F

Table 3.2: Average maximum muscle forces within phase 1 across speeds. The values represent the mean and standard deviation of the maximum muscle forces across participants during phase 1.

Muscle	Maximum Muscle Force (N): Phase 1		
	Slow	*SS*	*Fast*
Iliac	431.46±245.69	399.64±255.86	407.05±168.73
GlutMed	487.22±78.6	276.24±137.43	455.17±261.65
GlutMin	336.38±231.32	456.75±187.22	288.14±259.44
Semimem	81.57±45.96	147.26±196.41	198.58±170.67
RecFem	128.85±146.88	166.21±174.36	211.51±261.87
VasLat	698.39±253.82	723.95±330.86	767.05±249.66
VasInt	157.04±57.16	268.95±260.36	230.44±152.53
VasMed	236.68±88.41	275.11±96.66	342.63±226.34
GasMed	342.08±198.82	420.20±205.28	379.49±145.72
Sol	1634.52±407.32	1978.06±346.81	1894.81±476.00
PerLong	14.53±4.35	19.33±6.20	19.80±7.25
TibPost	23.40±5.59	30.40±8.42	30.74±9.63

Table 3.3: Average maximum muscle forces within phase 2 across speeds. The values represent the mean and standard deviation of the maximum muscle forces across participants during phase 2.

Muscle	Maximum Muscle Force (N): Phase 2		
	Slow	*SS*	*Fast*
Iliac	269.81±285.43	291.47±227.47	373.39±245.87
GlutMed	438.05±107.25	268.33±156.62	445.00±249.16
GlutMin	379.00±220.97	527.20±209.95	352.68±208.04
Semimem	87.19±48.55	182.73±247.45	161.71±170.51
RecFem	104.09±134.17	181.40±171.51	196.85±219.25
VasLat	646.87±336.95	926.69±354.64	1065.31±263.37
VasInt	146.92±79.69	483.92±433.90	313.20±115.76
VasMed	224.69±122.18	386.23±120.26	467.41±166.36
GasMed	188.62±75.13	235.32±86.23	208.65±74.10
Sol	1641.38±422.85	2189.66±557.39	1972.70±517.39
PerLong	16.36±5.01	89.74±181.03	23.53±6.24
TibPost	25.76±8.04	114.77±215.97	33.03±7.81

Table 3.4: Average maximum muscle forces within phase 3 across speeds. The values represent the mean and standard deviation of the maximum muscle forces across participants during phase 3.

Muscle	Maximum Muscle Force (N): Phase 3		
	Slow	*SS*	*Fast*
Iliac	497.86±210.77	621.44±55.63	611.39±160.93
GlutMed	497.59±209.67	265.60±187.45	362.64±178.76
GlutMin	494.62±185.28	530.63±73.48	422.03±242.64
Semimem	48.98±29.55	112.97±138.05	71.29±117.64
RecFem	246.69±215.36	266.00±156.31	369.92±235.18
VasLat	2198.53±371.13	1438.39±664.83	1465.89±559.54
VasInt	894.17±259.39	882.57±313.62	805.07±263.72
VasMed	1061.14±429.01	739.32±402.03	905.33±447.00
GasMed	284.68±262.01	367.32±206.29	364.36±187.59
Sol	2273.75±229.17	2202.48±378.50	2108.36±653.68
PerLong	374.05±279.37	406.84±243.63	469.42±224.91
TibPost	520.83±394.46	570.75±345.59	654.16±322.71

Table 3.5: Average maximum muscle forces within phase 4 across speeds. The values represent the mean and standard deviation of the maximum muscle forces across participants during phase 4.

Muscle	Maximum Muscle Force (N): Phase 4		
	Slow	*SS*	*Fast*
Iliac	239.14±78.76	220.25±46.31	338.65±48.40
GlutMed	157.61±41.99	150.29±63.32	181.57±66.25
GlutMin	25.11±15.78	48.42±40.08	42.97±27.58
Semimem	157.85±64.84	230.92±140.90	341.19±172.39
RecFem	104.79±51.74	104.83±64.11	147.83±77.85
VasLat	177.67±198.83	290.73±231.36	489.76±229.99
VasInt	31.84±50.54	63.70±51.82	109.69±56.57
VasMed	78.85±80.98	137.39±80.89	200.40±90.58
GasMed	130.19±41.96	135.83±85.93	148.08±76.77
Sol	89.01±156.35	273.26±216.75	279.97±200.55
PerLong	1.75±2.03	1.11±0.80	2.32±3.66
TibPost	2.20±2.87	1.33±1.03	3.16±5.15

3.4 Contributions of Muscles to Vertical Acceleration of the COM

There were significant differences in muscle contributions to vertical acceleration of the COM between muscles ($p<0.001$) and phases ($p<0.001$). There were also significant interactions between muscle and phase ($p<0.001$), speed and phase ($p<0.019$), and muscle and speed ($p<0.01$). Muscles have different contributions across phases and across speeds, but not within both phases and speeds. All other interactions were not significant. The soleus was the largest contributor to vertical acceleration across phases 1, 2, 3, and 4 for all speeds (5.87 ± 1.56 m/s^2, $p<0.001$) (Table 3.6). The vastus lateralis (VasLat) was the next largest contributor to vertical accelerations across phases 1, 2, and 3 for each speed (2.40 ± 0.75 m/s^2, $p<0.001$) (Table 3.6). Average maximum muscle contributions to vertical acceleration for the GlutMed, Iliac, Semimem, RecFem, VasInt, VasMed, GasMed, and GasLat are reported as these muscles produced an average contribution greater than 0.1 m/s^2 (Figures 3.18, 19, 20 and Tables 3.7, 8, 9). The Iliac and Semimem produced negative contributions to vertical accelerations while all other muscles produced positive contributions.

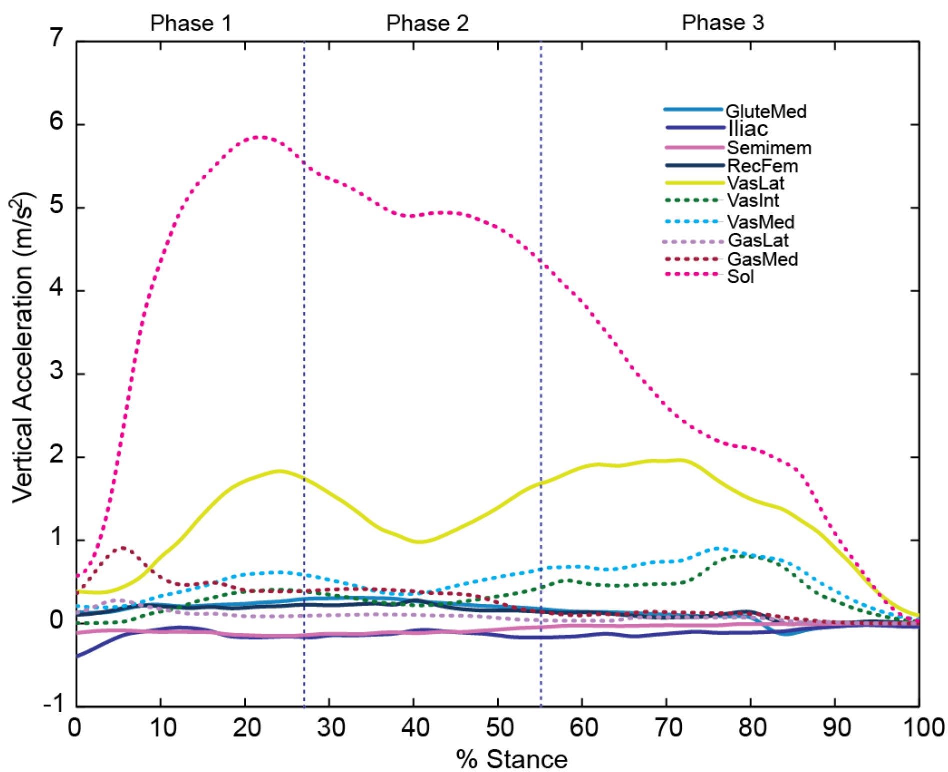

Figure 3.15: Waveform of average muscle contributions to vertical acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a slow speed.

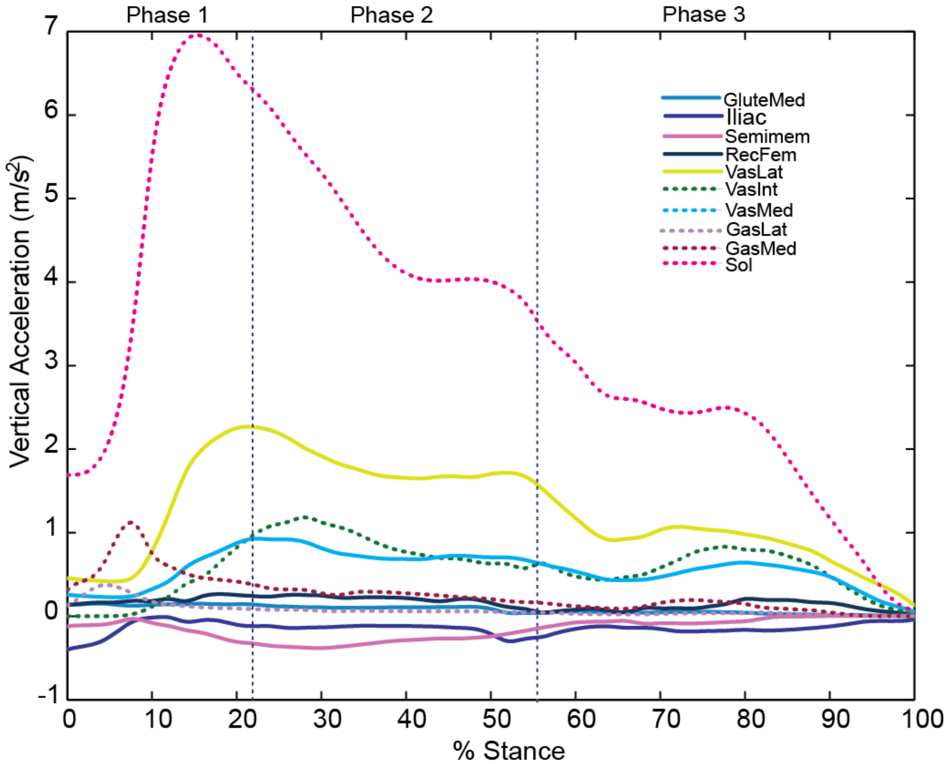

Figure 3.16: Waveform of average muscle contributions to vertical acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a SS speed.

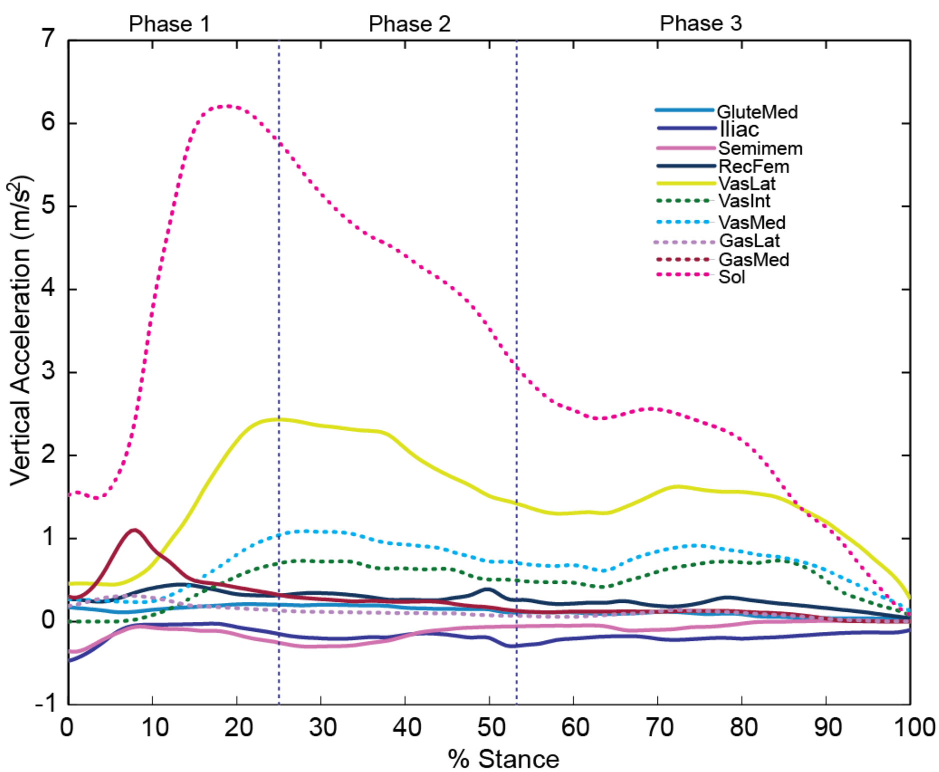

Figure 3.17: Waveform of average muscle contributions to vertical acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a fast speed.

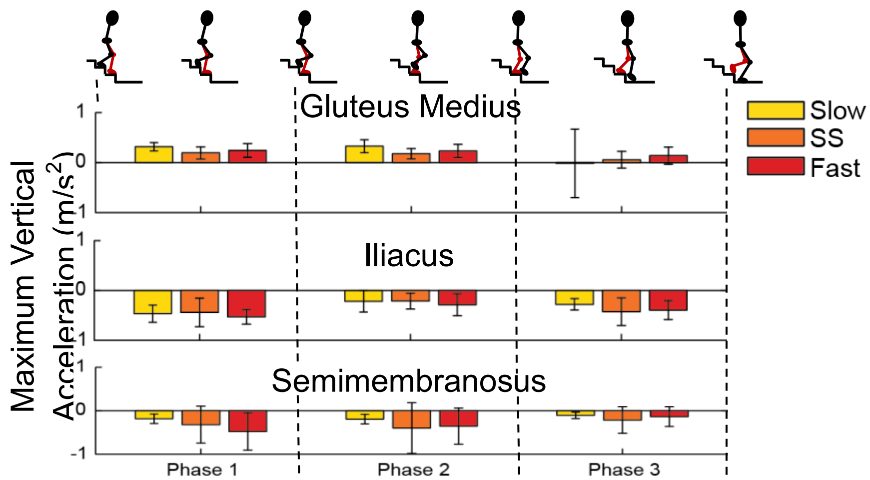

Figure 3.18: Average maximum muscle contributions to vertical acceleration of the COM for the gluteus medius, iliacus, and semimembranosus muscles for each speed within each phase of stance. Error bars span ± one standard deviation.

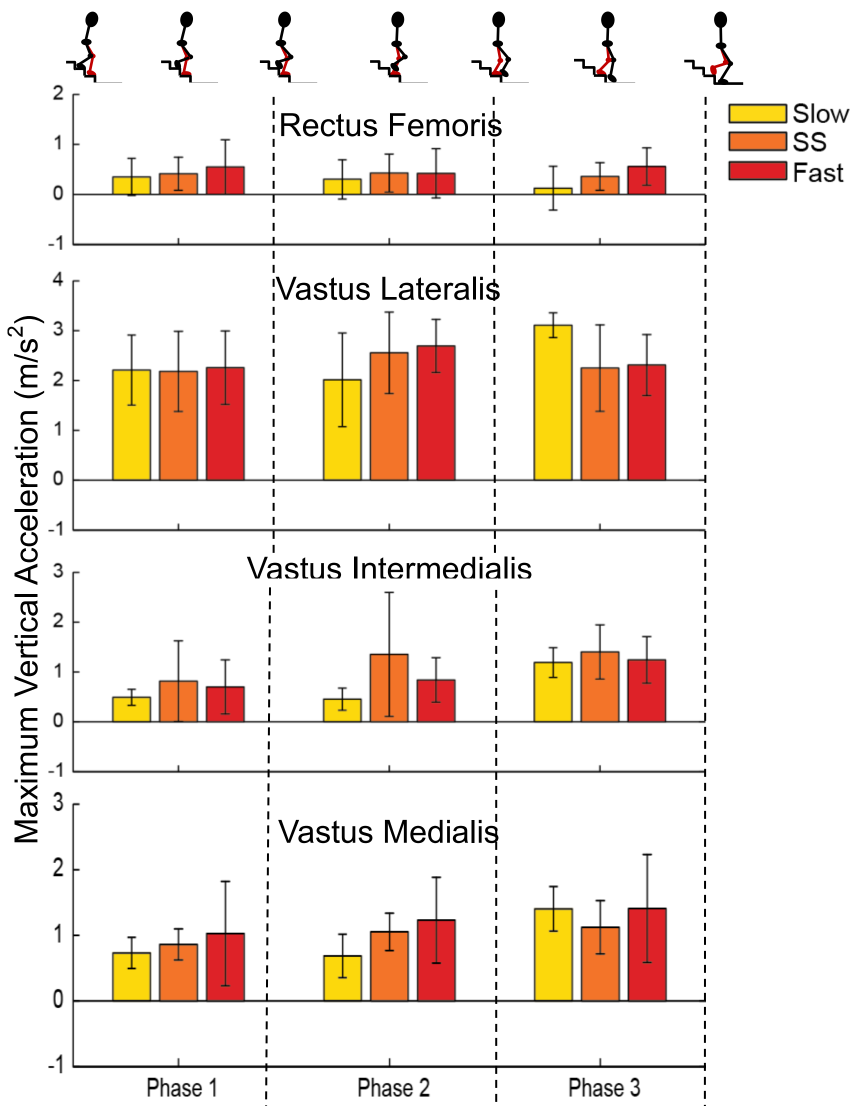

Figure 3.19: Average maximum muscle contributions to vertical acceleration of the COM for the quadriceps for each speed within each phase of stance. Error bars span ± one standard deviation.

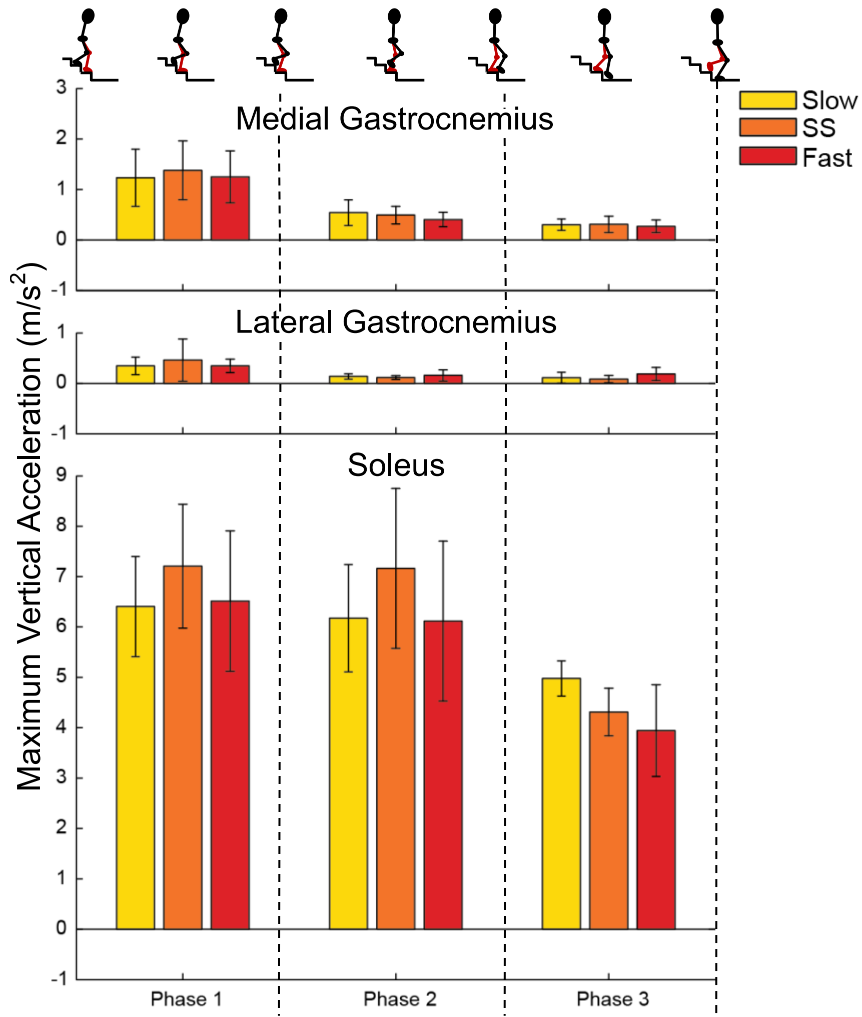

Figure 3.20: Average maximum muscle contributions to vertical acceleration of the COM for the posterior leg muscles for each speed within each phase of stance. Error bars span ± one standard deviation.

Table 3.6: Mean muscle vertical contributions across all speeds and all phases. The values represent the mean and standard deviation of all forces produced by a muscle. Means that do not share a letter are significantly different ($p<0.05$).

Muscle	Mean Muscle Force (N)	Grouping
Sol	5.84±1.56	A
VasLat	2.40±0.75	B
VasMed	1.06±0.54	C
VasInt	0.94±0.67	C D
GasMed	0.69±0.55	D
RecFem	0.39±0.40	E
GasLat	0.22±0.21	E
GlutMed	0.18±0.27	E
Iliac	-0.36±0.22	F
Semimem	-0.27±0.35	F

Table 3.7: Average maximum muscle contributions to vertical acceleration of the COM within phase 1 across speeds. The values represent the mean and standard deviation of the maximum muscle contributions to vertical acceleration of the COM provided by the respective muscle across participants during phase 1. A negative value indicates that the muscle opposed vertical acceleration of the COM.

Muscle	Maximum Contribution to Vertical Acceleration (m/s^2): Phase 1		
	Slow	*SS*	*Fast*
GlutMed	0.31±0.08	0.19±0.12	0.24±0.14
Iliac	-0.47±0.17	-0.44±0.28	-0.53±0.15
Semimem	-0.18±0.11	-0.32±0.43	-0.48±0.43
RecFem	0.35±0.37	0.41±0.33	0.55±0.54
VasLat	2.21±0.70	2.18±0.80	2.26±0.73
VasInt	0.49±0.16	0.82±0.81	0.70±0.54
VasMed	0.73±0.24	0.86±0.24	1.03±0.80
GasMed	1.23±0.57	1.38±0.58	1.25±0.51
GasLat	0.35±0.17	0.46±0.42	0.35±0.13
Sol	6.41±0.99	7.21±1.23	6.51±1.39

Table 3.8: Average maximum muscle contributions to vertical acceleration of the COM within phase 2 across speeds. The values represent the mean and standard deviation of the maximum muscle contributions to vertical acceleration of the COM provided by the respective muscle across participants during phase 2. A negative value indicates that the muscle opposed vertical acceleration of the COM.

Muscle	Maximum Contribution to Vertical Acceleration (m/s^2): Phase 2		
	Slow	*SS*	*Fast*
GlutMed	0.33±0.13	0.17±0.10	0.23±0.13
Iliac	-0.22±0.22	-0.21±0.16	-0.29±0.22
Semimem	-0.19±0.11	-0.40±0.58	-0.35±0.42
RecFem	0.30±0.39	0.43±0.38	0.42±0.49
VasLat	2.01±0.94	2.56±0.82	2.69±0.53
VasInt	0.45±0.22	1.35±1.24	0.84±0.45
VasMed	0.69±0.33	1.05±0.28	1.23±0.65
GasMed	0.54±0.25	0.49±0.17	0.41±0.14
GasLat	0.14±0.05	0.11±0.04	0.16±0.11
Sol	6.18±1.07	7.16±1.59	6.12±1.59

Table 3.9: Average maximum muscle contributions to vertical acceleration of the COM within phase 3 across speeds. The values represent the mean and standard deviation of the maximum muscle contributions to vertical acceleration of the COM provided by the respect.

Muscle	Maximum Contribution to Vertical Acceleration (m/s^2): Phase 3		
	Slow	*SS*	*Fast*
GlutMed	-0.02±0.68	0.06±0.17	0.14±0.17
Iliac	-0.28±0.11	-0.43±0.28	-0.39±0.19
Semimem	-0.11±0.08	-0.22±0.31	-0.14±0.23
RecFem	0.13±0.44	0.36±0.28	0.56±0.37
VasLat	3.11±0.25	2.25±0.87	2.31±0.61
VasInt	1.19±0.30	1.40±0.54	1.24±0.47
VasMed	1.40±0.34	1.12±0.41	1.41±0.82
GasMed	0.30±0.11	0.31±0.16	0.27±0.12
GasLat	0.11±0.11	0.08±0.07	0.19±0.13
Sol	4.98±0.35	4.31±0.47	3.94±0.91

3.5 Contributions of Muscles to Horizontal Acceleration of the COM

There were significant differences in muscle contributions to horizontal acceleration of the COM produced between muscles ($p<0.001$), speeds ($p<0.045$), and phases ($p<0.001$). There were also significant interactions between muscle and phase ($p<0.001$), muscle and speed ($p<0.001$), and muscle, speed, and phase ($p<0.003$). These outcomes indicate that the muscles provided significantly different accelerations from one another and across different phases and speeds of SD. The Sol was the largest contributor to horizontal acceleration across phases 1, 2, and 3 for all speeds ($1.33\pm0.66 m/s^2$, $p<0.001$) (Table 3.10). The VasLat largely opposed horizontal acceleration across phases 1, 2, and 3 for each speed (-0.83 ± 0.20 m/s^2, $p<0.001$) (Table 3.10). Across speeds, the Sol produced a significantly greater positive contribution to horizontal acceleration at fast speed than at slow speed during phase 1 ($p=0.0120$,) (Figure 3.18, Table 3.8) and during phase 2 ($p=0.0030$) (Figure 3.18, Table 3.11, 12). There were no additional significances across speeds. Average maximum contributions to horizontal accelerations for the GlutMed, GlutMin, Semimem, RecFem, VasInt, VasMed, GasMed, and GasLat are also reported as these muscles an average contribution greater than 0.03 m/s^2 (Figures 3.24, 25, 26 and Tables 3.11, 12, 13).

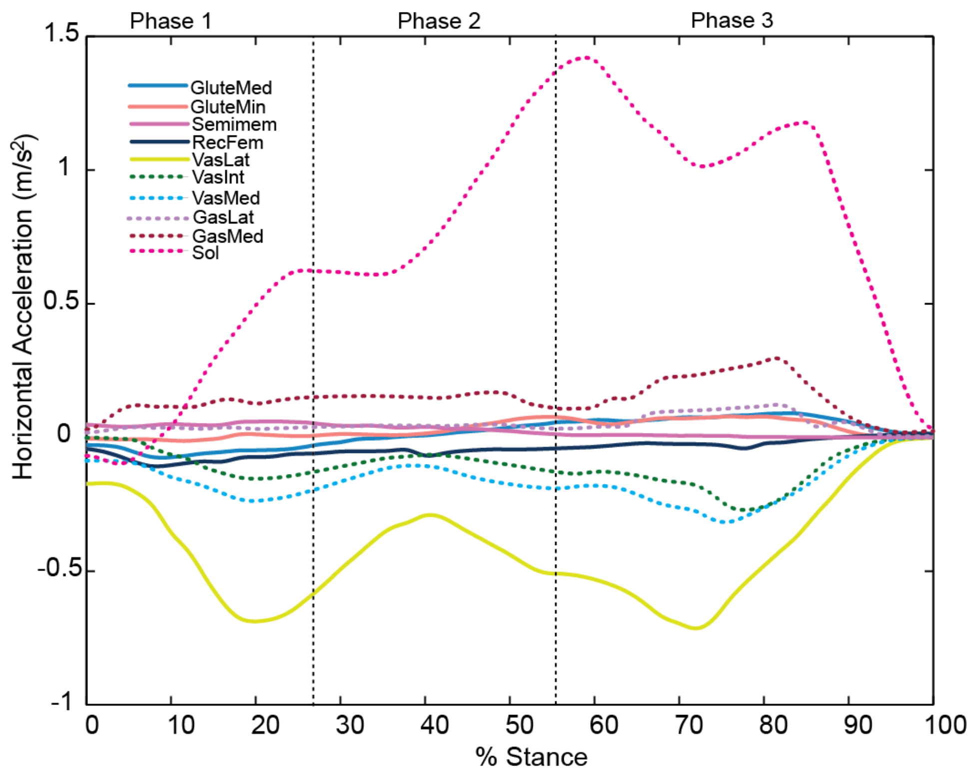

Figure 3.21: Waveform of average muscle contributions to horizontal acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a slow speed.

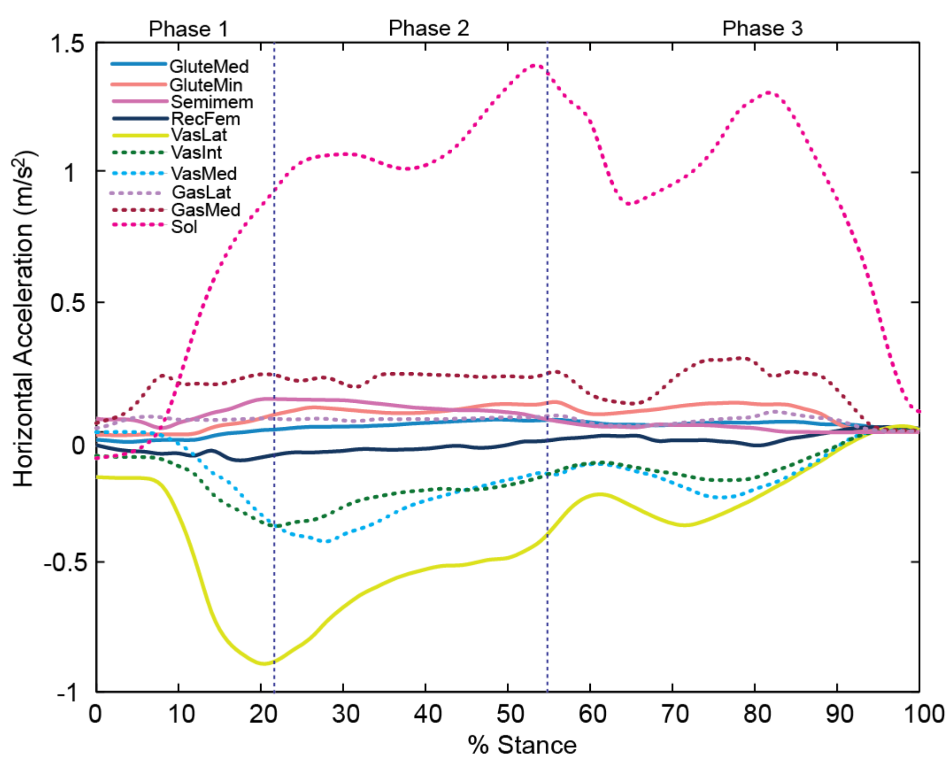

Figure 3.22: Waveform of average muscle contributions to horizontal acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a SS speed.

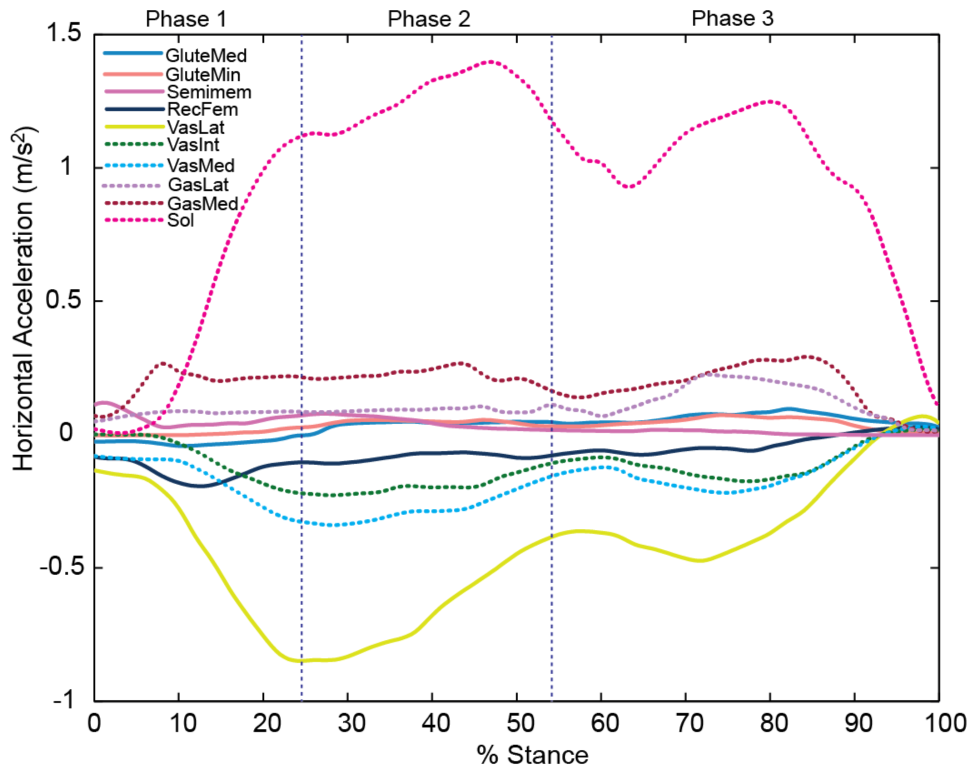

Figure 3.23: Waveform of average muscle contributions to horizontal acceleration of the COM across stance phase of one stair climbing cycle (SC) performed at a fast speed.

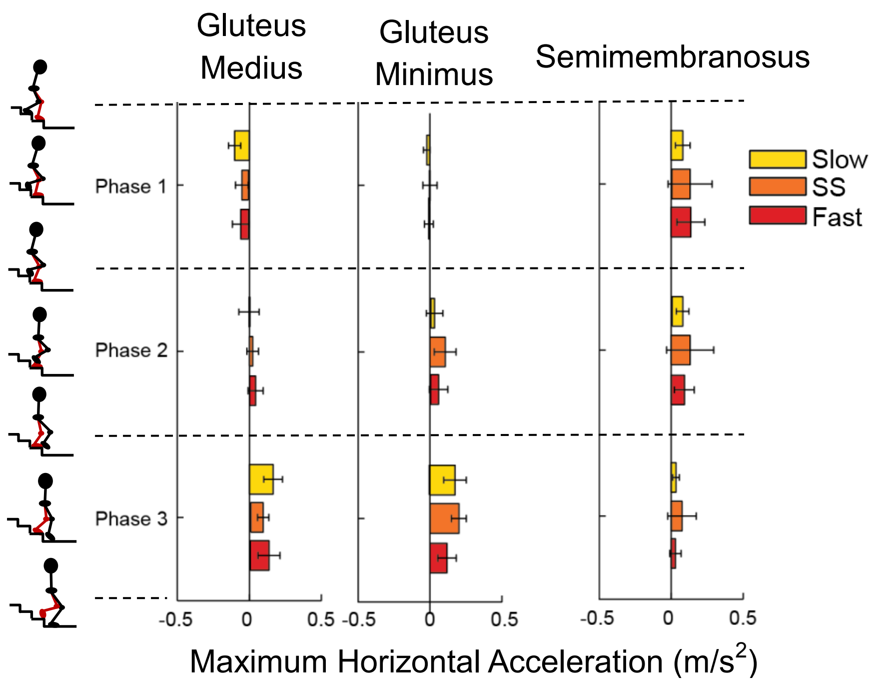

Figure 3.24: Average maximum muscle contributions to horizontal acceleration of the COM for the gluteus medius, gluteus minimus, and semimembranosus for each speed within each phase of stance. Error bars span ± one standard deviation.

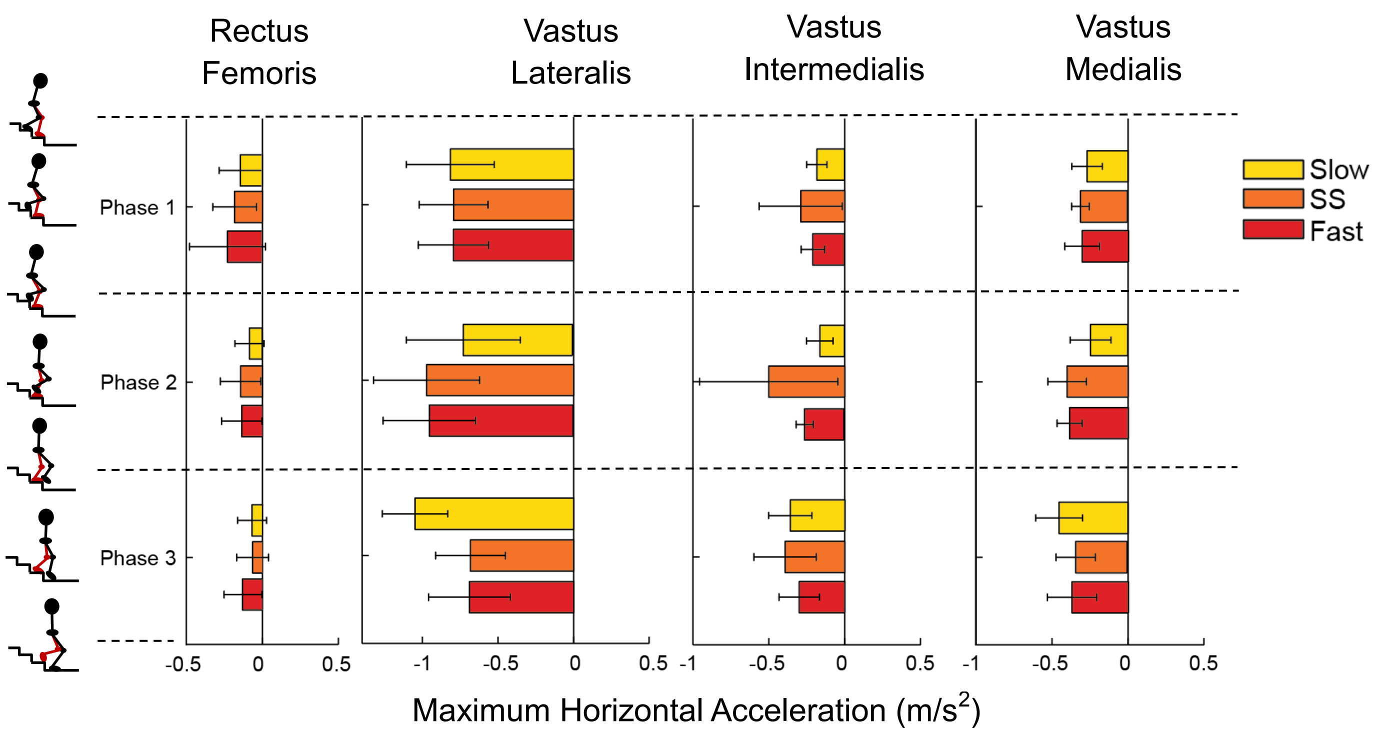

Figure 3.25: Average maximum muscle contributions to the horizontal acceleration of the COM for the quadriceps muscles for each speed within each phase of stance. Error bars span ± one standard deviation.

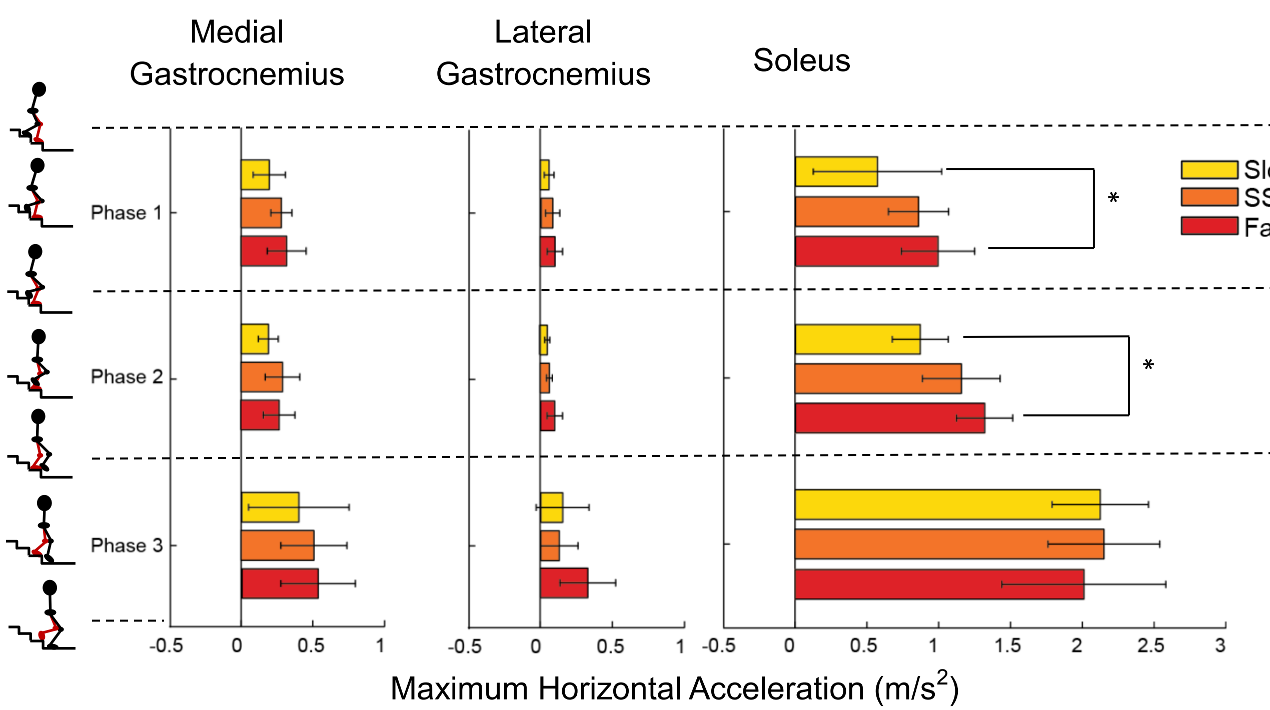

Figure 3.26: Average maximum muscle contributions to horizontal acceleration of the COM for the plantarflexors for each speed within each phase of stance. Error bars span ± one standard deviation. An asterisk (*) indicates that the contribution to horizontal acceleration provided by the muscle was significantly different between the respective speeds within the phase (p<0.0120).

Table 3.10: Mean muscle horizontal contributions across all speeds and all phases. The values represent the mean and standard deviation of all forces produced by a muscle. Means that do not share a letter are significantly different ($p<0.05$).

Muscle	Mean Muscle Force (N)	Grouping
Sol	1.34±0.66	A
GasMed	0.33±0.21	B
GasLat	0.12±0.13	C
Semimem	0.09±0.10	C
GlutMin	0.07±0.09	C
GlutMed	0.03±0.10	C
RecFem	-0.13±0.14	D
VasInt	-0.30±0.22	E
VasMed	-0.34±0.13	E
VasLat	-0.83±0.29	F

Table 3.11: Average maximum muscle contributions to horizontal acceleration of the COM within phase 1 across speeds. The values represent the mean and standard deviation of the maximum muscle contributions to horizontal acceleration across participants during phase 1. A negative value indicates that the muscle opposed horizontal acceleration of the COM.

Muscles	Maximum Contribution to Horizontal Acceleration (m/s^2): Phase 1		
	Slow	*SS*	*Fast*
GlutMed	-0.10±0.04	-0.04±0.04	-0.05±0.06
GlutMin	-0.02±0.02	0.00±0.05	-0.01±0.03
Semimem	0.08±0.05	0.13±0.15	0.14±0.10
RecFem	-0.14±0.14	-0.18±0.14	-0.22±0.25
VasLat	-0.82±0.29	-0.79±0.23	-0.79±0.23
VasInt	-0.18±0.07	-0.29±0.27	-0.21±0.08
VasMed	-0.27±0.10	-0.31±0.06	-0.30±0.11
GasMed	0.20±0.11	0.28±0.07	0.32±0.13
GasLat	0.06±0.03	0.08±0.05	0.10±0.05
Sol	0.57±0.45	0.86±0.21	0.99±0.26

Table 3.12: Average maximum muscle contributions to horizontal acceleration of the COM within phase 2 across speeds. The values represent the mean and standard deviation of the maximum muscle contributions to horizontal acceleration across participants during phase 2. A negative value indicates that the muscle opposed horizontal acceleration of the COM.

Muscle	Maximum Contribution to Horizontal Acceleration (m/s^2): Phase 2		
	Slow	*SS*	*Fast*
GlutMed	-0.01±0.07	0.02±0.04	0.04±0.05
GlutMin	0.03±0.06	0.11±0.08	0.06±0.06
Semimem	0.07±0.04	0.13±0.16	0.09±0.07
RecFem	-0.08±0.10	-0.14±0.13	-0.14±0.13
VasLat	-0.72±0.38	-0.97±0.35	-0.95±0.31
VasInt	-0.16±0.09	-0.50±0.46	-0.26±0.06
VasMed	-0.24±0.13	-0.40±0.13	-0.39±0.08
GasMed	0.19±0.07	0.29±0.12	0.27±0.11
GasLat	0.05±0.02	0.06±0.02	0.10±0.05
Sol	0.87±0.19	1.16±0.27	1.32±0.20

Table 3.13: Average maximum muscle contributions to horizontal acceleration to the COM within phase 3. The values represent the mean and standard deviation of the maximum muscle contributions to horizontal acceleration across participants during phase 3. A negative value indicates that the muscle opposed horizontal acceleration of the COM.

Muscle	Maximum Contribution to Horizontal Acceleration (m/s^2): Phase 3		
	Slow	*SS*	*Fast*
GlutMed	0.16±0.07	0.09±0.04	0.13±0.08
GlutMin	0.18±0.08	0.20±0.05	0.12±0.06
Semimem	0.03±0.02	0.07±0.10	0.03±0.04
RecFem	-0.06±0.09	-0.06±0.10	-0.13±0.12
VasLat	-1.05±0.22	-0.68±0.23	-0.69±0.27
VasInt	-0.36±0.14	-0.39±0.21	-0.30±0.13
VasMed	-0.45±0.15	-0.34±0.13	-0.37±0.16
GasMed	0.40±0.18	0.51±0.13	0.53±0.19
GasLat	0.15±0.35	0.13±0.23	0.33±0.26
Sol	2.12±0.34	2.15±0.39	2.01±0.57

4 Discussion

To our knowledge this is the first study to estimate muscle forces and their contributions to the acceleration of the COM during SD at different speeds using three-dimensional dynamic simulations. Due to the relatively small number of subjects, there were only a limited number of significant differences found for a certain muscle across speeds within a phase. Based on the results that were found across speeds, I reject my hypothesis that as descending speed increases, muscle forces from the quadriceps would increase in a young, healthy population. This is because the VasLat produced a significantly greater force at slow speed than at SS or fast speed in phase 3. Because of the lack of significant results, I also reject my hypothesis that as descending speed increases, contributions to the vertical and horizontal acceleration of the COM from the quadriceps and both muscle forces and contributions to the horizontal and vertical acceleration of the COM from the gluteal muscles and plantarflexors would increase in a young, healthy population. Even though not statistically significant, there were additional trends observed for muscle forces and muscle contributions to acceleration across speeds that will be discussed below.

4.1 Muscle Forces and their Contributions to Acceleration of the COM

The results from this study compared favorably with a study conducted by Lin et al. (2014) that used three-dimensional models to estimate muscle forces and their contributions to acceleration of the COM during SD [11]; noting that this study did not report numerical values for muscle force, only patterns of muscle force are compared below. In agreement with Lin et al., both studies showed that the Sol produced the largest force during stance phase, having similar magnitudes for SS speed. The vasti (VasLat, VasMed, VasInt) are shown to produce the second largest force during all phases by this study, whereas with Lin et al., the GlutMed produced the second largest force during SD. The GlutMed is shown to produce a larger force than the vasti in the first half of stance, where as in our study the GlutMed produces less force than the VasLat in all phases.

Lin et al. also investigated muscle contributions to vertical and horizontal acceleration during SD at a SS speed and found that the Sol was the largest contributor to vertical and horizontal acceleration of the COM in the first and second half of stance phase and that its contribution was smaller in the second half, which agrees with this study's results. However, the Sol opposed horizontal acceleration in the first after of stance in Lin et al.'s study, while the results from this study suggested that it induced forward acceleration (positive horizontal acceleration). Both studies also found that the vasti were the second largest contributors. However, the results did not agree between studies with regard to contributions from the gluteal muscles. Our study found that the gluteus maximus produced a contribution less than 0.1 m/s^2 and the gluteus medius produced a contribution of less than 0.19 m/s^2 while their study found a contribution of approximately 2 m/s^2 each for the gluteus maximus and medius.

These differences between studies may be due to several reasons. One difference may be in the calculation of muscle activations. While both studies used static optimization to calculate muscle activations and forces, there were bounds applied to the calculated muscle activations for those muscles that had experimental EMG in this study. However, placing bounds on the calculated activations allowed for better agreement between the simulation and the EMG [25]. Furthermore, the model used in this study, the Full Body Model 2015 has 96 muscles and 46 degrees of freedom while the model used in Lin et al.'s study, Gait2392, has 92 muscles and 23 degrees of freedom. Due to the differences in number of muscles and since the objective function of SO was minimizing the sum of muscle activations squared; having more muscles will cause differences in calculated muscle activations and forces. Since the Full Body Model 2015 has more degrees of freedom, the number of coordinates tracked by IK is higher, which could result in slightly different kinematics than if the model were to have fewer degrees of freedom like the Gait2392 model (i.e. no flexible back or arms). Having these different kinematics could lead to different calculated muscle forces as the muscle would have a different position on the force-length-velocity curve. There were also differences in participants tested as well as the self-selected speed. In this study, there were eight subjects, three males and five females that had an average age of 22±1.5 years whose average SS SD speed was 0.65 m/s whereas in Lin et al.'s study, there were fifteen subjects, four males and eleven females that had an average age of 54±8 years whose average SS SD speed was 0.74 m/s.

It is believed that this is the first study to investigate muscle forces and contributions to acceleration at different speeds. Due to the lack of significance, many of the following patterns are only trends seen and interpreted from the results. In phase 4, the muscle forces from all muscles were smaller than in all other phases and all seemed to increase with increasing speed. This is expected because the limb in not loaded during swing phase. The muscle forces produced by quadriceps, Iliac, and Semimem seem to increase with increasing speed for most phases of SD while the forces from TibPost and PerLong also seemed to, but only in phase 3. One surprising finding is the force from the VasLat in phase 3 at slow speed was significantly greater than force at SS or fast speed. Additionally, in phase 3, the VasMed and Sol also show trends of decreasing force with increasing speed. It should be noted that these three muscles (two quadriceps, one plantarflexor) produce their maximum force during phase 3 of SD and this maximum is decreasing, not increasing with speed. It is during this phase of SD that these three muscles are assisting in lowering the body towards the next stair, indicating that less VasLat, VasMed, and Sol muscle force may be needed to lower one's COM while descending faster. Other factors, such as joint contact forces, may increase with increasing speed to counteract gravity and prevent falling, so the lower muscle forces found in this study may not necessarily indicate that descending stairs at a faster speed is easier.

The muscle contributions to vertical acceleration of the COM from the Iliac and Semimem did not change with speed and were negative, which means they assisted in lowering the body's COM down the stairs. Additionally, the contributions from the GlutMed, GasMed, VasInt, and GasLat seemed to remain constant within a phase across speeds and were positive. The quadriceps, except for the VasInt, contributed positively to vertical acceleration and all seemed to increase with increasing speed, but again this pattern was not seen for the VasLat and Sol in phase 3. In phase 3, the VasLat contribution to vertical acceleration seemed to decrease from slow to SS speed and the Sol contribution to vertical acceleration also seemed to decrease with increasing speed. Therefore, as one descends faster, the VasLat and Sol seem to provide less vertical support during controlled lowering.

The muscle contributions in the horizontal direction were positive for the GlutMin, Semimem, GasMed, GasLat, and Sol, which indicates that these muscles propel the body forward while horizontal contributions from the quadriceps (vasti and RecFem) were negative, which means these muscles were opposing the forward motion of the body to possibly prevent falling forward. Furthermore, the contributions to horizontal acceleration from the quadriceps did not seem to increase or decrease with increasing speed, except for in phase 3, in which, the VasLat contribution to horizontal acceleration at slow speed was larger than its contributions to SS and fast seeds. Interestingly, this is not consistent with the pattern observed for the quadriceps' muscle forces and contributions to vertical acceleration as they seemed to increase with increasing speed, except for in phase 3. These larger horizontal contributions produced by the quadriceps at a slow speed possibly mean that the slower one descends stairs, the more resistance is needed to stop the body from falling forward. In contrast to the quadriceps, the Sol did not continue the trend of decreasing contribution with increasing speed in phase 3. Instead, the horizontal contributions from the Sol seem to increase with increasing speed in all phases. This indicates that the Sol helps propel the body forward more at faster speeds. Contributions from the GasMed and GasLat also seemed to increase with increasing speed in all phases. Therefore, the Sol, GasMed, and GasLat are needed to increase propulsion at faster speeds, which allows one to descend faster, while the quadriceps are needed to oppose this motion to stop one from moving too fast and to possibly prevent forward falling. It should also be noted that vertical and horizontal COM acceleration were not only dependent on contributions from muscles, but were also influenced by resistance to gravity provided by skeletal alignment (Figures 2.12 – 2.14). This is especially true of vertical acceleration through the stair climbing cycle, in which skeletal alignment seems to act to keep the COM upright and to resist gravity, which is consist with previous studies that have examined muscle-induced accelerations during gait [14, 16]. Compared to walking, SD has a larger vertical acceleration of the COM that is largely muscle-induced in addition to gravity [16, 22]. However, for horizontal accelerations, SD has a smaller horizontal acceleration of the COM in comparison to walking. This indicates that the demands placed on the muscles during SD are to more-so to help support the body rather than propel it forward [16].

It was not expected that the forces and contributions to vertical acceleration of the COM from the Sol, muscle forces from the VasMed, and forces and contributions to vertical and horizontal acceleration of the COM from the VasLat would decrease with increasing speed for phase 3. This was because Liu at al. (2008) found that during increasing walking speeds, contributions from all muscles generally increased with increasing speed, with especially large increases in contributions from the vasti between slow and SS walking speed [14]. The patterns seen here, while surprising, can be linked to Lewis et al. (2015)'s study [9]. The VasLat and VasMed pattern can be directly related to the maximum knee joint torque not increasing with increasing speed since the vasti are knee flexors; the Sol pattern can be related to the maximum ankle joint torque not increasing with increasing speed since the Sol is a plantarflexor. The muscle force patterns for the VasLat and VasMed are particularly interesting because quadriceps weakness has been shown to increase the risk of falling [29], slower walking speeds [30], and make stair climbing more difficult [30]. However, since the maximum forces and contributions to vertical and horizontal acceleration from the VasLat decreased with increased walking speed, quadriceps weakness may not be a limiting factor in how fast a young, healthy individual can descend stairs.

The results from this study were verified in multiple ways. The SO muscle activations followed similar patterns to the experimentally collected EMG (Figure 2.5, 6, 7). The average joint torques calculated from RRA matched very well to those found by multiplying the muscle force from SO by the moment of the corresponding muscle (Figure 2.8, 9, 10) meaning that reserve actuators were not contributing largely to the SD motion.

4.2 Limitations

Several limitations should be considered. One limitation of this study is that the muscle activations calculated from SO do not match the experimentally collected EMG (Figure 2.5, 6, 7) exactly. In an attempt to better replicate the experimental EMG, we tried implementing another method of static optimization, developed by Joe Ewing, a PhD candidate in the lab, which tracks the experimental EMG while also tracking the joint torques from RRA. It penalizes solutions that diverge from the experimental EMG and the RRA joint torques instead of just trying to minimize the sum of muscle activations squared. However, after several attempts of trying to implement this method, it was not used for this study because some of the muscles for some subjects' trials would exhibit physiologically impossible spikes in the force due to the residuals at these points being extremely high. Due to time and resources, I decided to use the SO OpenSim tool instead, constraining the calculated muscle activations with upper and lower bounds so that they more closely followed the experimental EMG. While there are still small differences between the muscle activations from SO and the experimentally collected EMG even after applying these bounds, it is believed that this approach is an appropriate first step in evaluating muscle forces and their contributions to acceleration during SD across differing speeds. Future work will entail modifying this method so that it can be used for this and other stair related studies.

Furthermore, since the results from this study are for a young, healthy population, they cannot be assumed to be representative of the other populations like those who have difficulty performing the task [31, 32]. However, by testing a young, healthy population, these results build a baseline for future studies from which compensatory strategies and deficits can be identified in elderly, pathological populations, and others who have difficulty while descending stairs. While we had a small sample size that was similar to other simulation-based studies [14], many of the patterns for muscle force and contributions to acceleration of the COM seen across speeds were not statistically significant. Future steps include adding more subjects to increase statistical power.

5 Conclusions

5.1 Contributions

Stair descent is a difficult and potentially dangerous task for the elderly and those with pathologies. However, rehabilitation is not 100% effective for the populations that have difficulty. Using dynamic simulations to investigate SD can help to further understand how populations descend stairs, including how speed effects the mechanisms involved in SD. Previous studies have not looked at how muscle forces and their contributions to vertical and horizontal acceleration of the COM affect SD across speeds. A custom three-dimensional musculoskeletal model and dynamic simulations were used to investigate muscle force and their contributions to acceleration in a young, healthy population at different speeds. The results showed that the muscle forces and contributions to vertical acceleration of the COM from the Sol, muscle forces from the VasMed, and muscle forces and contributions to vertical and horizontal acceleration of the COM from the VasLat decreased with increasing speed in phase 3(controlled lowering) of SD. It is important to note that the maximums for the Sol, VasMed, and VasLat occur in phase 3, but these maximums seem to decrease with speed, not increase. These patterns in muscle force and contribution to acceleration helped to better understand why the joint torque from the hip, but not the knee or ankle, increases with increasing SD speed in a young, healthy population as reported by Lewis et al. [9]. Furthermore, muscle strengthening, particularly quadriceps strengthening, is a common rehabilitation technique used for populations is used to improve physical function for these patients that have difficulty completing tasks like SD. Since the results from this study suggest that the VasLat may not be limiting how fast a person can descend stairs, this technique may not be the most effective rehabilitation technique to use. Therefore, further investigation needs to be done to develop other rehabilitation techniques that could help to improve patient performance during SD.

5.2 Future Work

Additional work needs to be done with more subjects to better understand these patterns and to increase statistical power. Next, older, healthy and pathological populations can be investigated to understand the mechanisms they use during SD, particularly with regard to muscle forces and their contributions to the acceleration of the COM. When comparing these results between such populations, this study will act as a baseline to identify strategies and deficits in those populations, which may eventually lead to improved rehabilitation for the elderly and pathological populations and improve their performance during difficult tasks like SD.

5.3 Summary

Dynamic simulations of young, healthy individuals were used to estimate muscle forces and their contributions to acceleration of the COM during SD performed at different speeds. Certain muscle forces and contributions to acceleration decreased with increasing speed, which helped explain unexpected joint torques seen in a previous SD study. Understanding this pattern and comparing it to other populations' patterns during SD may lead to improved rehabilitation for pathological populations in the future.

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